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# Estimating and Valuing Morbidity in a Policy Context Proceedings of June 1989 AERE (Association of Environmental and Resource Economists) Workshop. Held in Research Triangle Park, North Carolina on June 8-9, 1989

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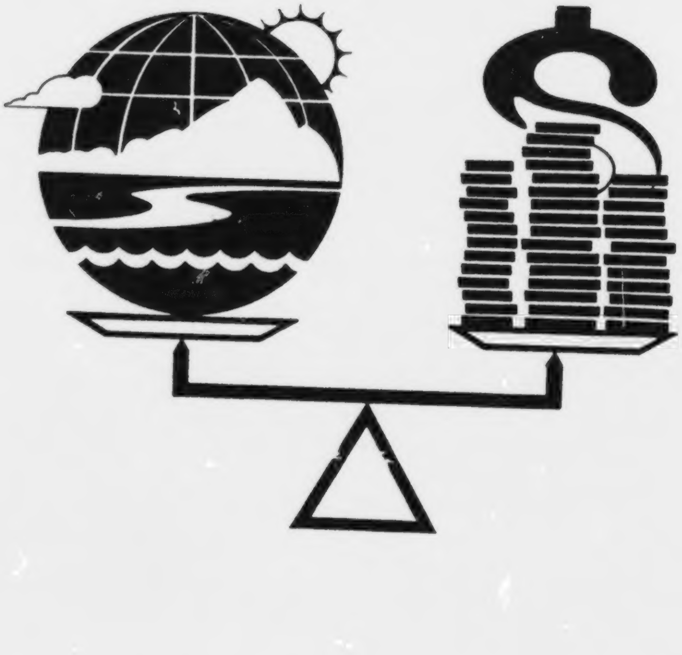

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AERE Workshop

# Estimating and Valuing Morbidity in a Policy Context

# Proceedings

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# THE ROLE OR EPIDEMIOLOGY IN DEVELOPING USEFUL DATA FOR PUBLIC HEALTH POLICY

by

Daniel A. Hoffman

# The Role of Epidemiology in Developing Useful Data for Public Health Policy

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## INTRODUCTION

In the last two decades, the role of epidemiology in providing data for public health policy makers has become more prominent than any time in the history of this discipline. Its greatest advantage is that it provides direct human evidence of the health outcomes from various environmental exposurs, unlike animal models. However, there are many caveats that need to be attached to these data. The objective of this paper is to review some of the basic limitations to the epidemiologic method, both in study design and in interpretation of the data. The perspective that I will present is that of an epidemiologist in a public health agency. The Centers for Disease Control does not engage in the development of regulations or have a large program in risk assessment.

Our principle function is to serve the state and local health departments by offering advice and assistance when necessary through field investigations of potential public health problems-in our case, environmentally-induced disease. This involves the identification of study hypotheses, designing the study, developing the necessary survey instruments, collecting health data, analyzing this data, and finally offering our interpretation and recommendations. Basically we engage in the classical epidemiologic method of hypothesis generation and testing through field investigations. Consequently, my talk today is focused on the techniques involved in acquiring these data, and examining the strengths and weaknesses of these data as they relate to various interpretations of their meaning for use in risk assessments, regulatory actions, or public health policy decisions.

## DESCRIPTION OF EPIDEMIOLOGIC METHODS

The assessment of effects on humans of various environmental exposures relies heavily upon the results from testing of animal models and clinical and epidemiological studies. However, the most important advantage that epidemiological studies have over animal investigations is that they provide direct evidence of the effects of toxic exposures in humans. Conversely, human studies are difficult to conduct properly and the interpretation of the results from these studies makes life difficult for both regulators and policy makers. Part of the problem in interpretation stems from the design of these studies which can be very complex. Another problem is dealing with the inherent biases which inevitably creep into the interpretation of the data, no matter how thoroughly these have been addressed either in the study design or analysis. This stems from the fact that, with the execption of clinical trials, epidemiological studies are observational by nature, not experimental. Not only do humans vary widely in their response to toxic agents but they vary also in their capacity for response as well as in their exposure to factors such as alcohol and tobacco, which may may greatly modify the nature or severity of their responses to toxic exposures. One example would by the relation between radon exposure and cigarette smoking which could either be additive, submultiplicative or mulplicative depending upon which data is reviewed and which model is applied to that data.

Despite these difficulties, techniques for the evaluation of data from human studies have been developed and refined. The epidemiological method has matured to the point that it has withstood the criticism that it is incapable of establishing the etiology of disease. Epidemiological inferences have been sustained and corroborated by the results of toxicological and biochemical studies, and epidemiology has proven to be a powerful tool for the exploration of both qualitative and quantitative cause-and-effect relationships between environmental exposures and human disease. However, there is still much to be done, especially at the rather low levels of exposures that most human populations experience, to further refine the tools of epidemiology. I would now like to briefly discuss some of the various study designs used in epidemiology. Next I will address some of the sources of bias in epidemiologic data, and conclude with a discussion on interpretations of causality based on data derived from epidemiological studies. Two areas of study which I will not discuss in any detail today are the appropiateness of animals models as they apply to risks in humans and the use of biomarkers as indicators of risk in epidemiologic studies. The majority of our experience at CDC has been concerned with collection and interpretation of epidemiologic data so the principal focus of my talk will be on that process.

The most commonly used designs in epidemiology are: 1) case reports; 2) ecological or correlational studies; 3) cross-sectional studies; 4) case-control studies; and 5) cohort studies.

## 1. CASE REPORTS

Çase reports identify one or more cases of a disease that have been detected by clinicians, by company or union officials, or by through active surveillance or passive reporting such as cancer registeries. The first recorded case studies of environmental disease were Sir Percival Pott's observations of scrotal cancer among chimney sweeps in London. Publication of such case reports often constitutes the first recognition that a problem of environmnetally induced disease exists, and subsequent epidemiological assessment proceeds from this recognition. A more recent example includes the first recorded cases of AIDS by clinicians at UCLA medical center in 1978. In a case series, an inference of causal association between causation and an environmental agent is based on the plausability of the following considerations: clustering of the cases in a limited time frame; the relative rarity of the types of diseases observed; a history of common environmental exposure; an the apparent strength of the association. The most common use of case reports are hypothesis generation, surveillance, and case registries

## Surveillance

The case report has historically been an important surveillance tool, especially for recognition of infectious diseases. Occupational case reporting has been useful in terms of reporting occupational injuries for workman's compensation, but not so much for occupational diseases due to the long latency period between exposures and disease. A more recent use of case reports as a surveillance tool is for the identification of senital health events. These are cases of disease associated with well-characterized causes whose appearance signals a breakdown in mechanisms for disease prevention. This method has been applied with success in the reduction of maternal and infant mortality and has been extended to such environmental illnesses as lead poisoning.

## Case Registries

Other surveillance systems relying on case reports include case registries, such as the CDC Dioxin Registry or workers suspected of having been exposed to dioxin. These exposure registries perform the task of grouping potentially high-risk populations for future epidemiological studies.

An advantage of case reports over most other types of epidemiological studies is their low cost. In addition, a short lag time between identification of cases and dissemination of information is more typical of case reports. However, relying on case reports as an early warning system is less useful when:

1) the cases are sporadic;
2) the relative risk is low;
3) the outcome is a common disease or a symptom with multiple common etiologies such as lung cancer or heart disease;
4) there is a long latent period between exposure and effect; and
5) there is a continuum of disease and health and no clear distinction between cases and noncases is possible, for example, premalignant dysplasia and carcinoma in situ.

In addition, case reports can provide only a rough estimate of disease frequency, in that they give no information on the size of the population at risk and thus make it impossible to calculate a disease rate. Finally, case reports are difficult to generalize to a population since the population from which the cases are identified is not usually well defined.

## 2. CORRELATIONAL OR ECOLOGIC STUDIES

Another type of descriptive tool used by epidemiologists is the so-called correlational or ecologic study, which uses data from entire populations to compare disease frequencies between different groups during the same period of time or in the same population at different points in time.

As an example of the former, correlational studies have suggested that various dietary components, in this case per capita meat consumption, may be risk factors for colon cancer. Figure 1 shows the correlation between per capita consumption of meat and rates of colon cancer in women from a large number of countries. As apparent from this figure, the rates of colon cancer are lowest in countries with the lowest per capita meat intake and vice versa.

Figure 2 illustrates the change in disease frequency within the same population over time. In this slide, the difference between tge approximately 820,000 deaths from coronary heart disease that would have been expected in the United States if the 1968 rates had continued to apply and the approximately 630,000 deayhs actually observed. Such data suggest two possible explanations: 1) that the decline in deaths from coronary heart disease could be due to prevention due to improvements in life-style habits and consequent risk factor reduction, and 2) that while the rates of CHD did not decline, persons were surviving longer due to improvements in medical management of CHD.

While correlational studies are useful in developing hypotheses for study, they cannot be used to test them because of a number of imitations inherent in their design.

1) Correlational studies refer to populations rather than to individuals. Therefore, it is not possible to link an exposure to occurrence of disease in the same person.

2) The distribution of other risk factors which may account for different rates of a disease, may be differentially distributed among populations. This is known as the "ecologic fallacy".

## 3. CROSS-SECTIONAL STUDIES

Another type of descriptive study design is the cross-sectional survey, in which the status of an individual with respect to the presence or absence of both exposure and disease is assessed at the same point in time. For example, the Health Interview Survey is a national cross-sectional study that periodically collects extensive information by questionnaire from a sample of over 100,000 persons throughout the United States. These studies often rely on personal interviews or questionnaires to obtain demographic information, symptomatic, and exposure data on clinical evaluations based on physical examinations and laboratory and environmental sampling data to identify the characteristics of the sample population and to quantitate exposure to potential risk factors. An advantage of the cross-sectional survey is the rapid estimation of numerator values for determining frequency or prevalence rates of both exposure and effects. Limitations of this method include the inability to distinguish whether the exposure preceeded the development of disease or whether the presence of disease affected the individual's level of exposure, since exposure and disease are assessed at the same point in time. Cross-sectional approaches have limited usefulness in cancer studies because of the usual low prevalence of cases. It is also extremely difficult to quantify exposure in cross-sectional studies. However, for factors that remain unaltered over time, such as sex, race or blood group, the cross-sectional survey can provide evidence of valid associations.

Five common pitfalls can be found in the cross-sectional method. These are:

1) Selection bias, in that a nonrepresentative sample of the population may be surveyed, limiting the generalizability of the survey results;

2) Confounding bias, which can result for factors related to both exposure and outcome, such as age;
3) Inadequate sensitivity of the survey instruments. This includes specificity, which is the ability to detect "true" negatives, and sensitivity or the ability to detect "true" positives;
4) Lack of standardization of the instruments used for data collection, which may prohibit the pooling of data from multiple surveys; and
5) Inadequate validation of either exposures of health outcomes, resulting in misclassification of either category.

Summarizing, in general, cross-sectional studies are useful for raising the question of the presence of an association rather than testing a hypothesis.

The next two types of epidemiologic studies are observational in design. These are the case-control study and the cohort study.

In theory, it is possible to test a hypothesis using either design strategy. In practice, however, each design offers certain unique advantages and disadvantages. In general, the decision to use a particular design is based on the features of the exposure and disease, the current state of knowledge, and logistic considerations such as available time and resources.

## 4. CASE-CONTROL STUDIES

In the case-control study, a case group or series of patients who have a disease of interest and a control or comparison group of individuals without the disease are selected for investigation, and the proportions with the exposure of interest in each group are compared. Lung cancer patients, for example, can be compared to persons without that disease for differences in exposures, such as cigarette smoking, occupational exposures, and radon levels in the home. The relative frequency of distribution of the exposure in the case and control groups is usually evaluated by computing an odds ratio which is defined as the product of the number of exposed cases and unexposed controls divided by the product of the unexposed cases and exposed controls. This is also somtimes known as the cross-product odds ratio because of the manner in which it is calculated.

Case-control studies can be conducted relatively rapidly. Many simultaneous exposures can be evaluated in relation to even the rarest disease. Howver, the sequence of exposure-health event is often difficult to assess if the case population includes patients selected from historical records. If the disease studied is rapidly fatal, interviews with surrogate respondents may be required which may result in misclassification of exposures. The individual exposure status is often difficult to quantify with any precision, especially in environmental studies, and control of possible confounders may require a complex design or analysis. Consequently, only environmental exposures with a high prevalence and relative strong toxic effect are effectively studied by the case-control method.

## 5. COHORT STUDIES

In a cohort or follow-up study, the study population is divided on the basis of exposure status. For example, in a recent study of the health effects of volatile organic compounds in Michigan, we assembled study cohorts on the basis of whether or not VOC's were detected in their well water and if they had lived for a specified period of time in the study area. Residents who had moved away prior to the initiation of the study were still eligible for inclusion in either the exposed or unexposed cohorts. Once the exposure status of the study cohorts has been determined, which is sometimes quite complex and can result in misclassification of exposure status thus biasing the study outcome towards the null hypothesis of finding no effect, the history of disease is determined in both the exposed and unexposed groups. The rate of disease in the exposed group is compared to that in the unexposed group resulting in a relative risk of disease which could be due to the exposure being studied. This is also called the rate ratio since it is simply the ratio of two incidence rates. Both of these measures of association include a factor for follow-up time known as the person-year. This is simply defined as the interval from the time exposure began to the date of diagnosis of disease, death, loss-to-follow-up or, if disease-free, an arbitrary date.

The strengths of the cohort approach include the following:

1) the sequence of exposure and health outcome can be studied;

) many health outcomes can be evaluated with regard to the one exposure of interest (although this may have become a problem in some studies as multiple comparisons inevitably lead to at least one "significant" finding);

3) the initial exposures can be quantified through historical records or even more so if there is a

biologic marker of exposure such as blood or bone lead levels;

4) rare exposures can be studied;

5) collection and analysis of potential confounding factor is possible; and

6) absolute risks may be calculated for use in public health prevention strategies.

Some of the drawbacks to the cohort approach are the expense and difficult logistics of these studies, the potential for misclassification of exposure and disease outcome resulting in a biased estinate of risk, and the inability to study rare disease because of the very large populations necessary for study. This latter drawback is important in studying the effects of low-level environmental exposures. Because the anticipated risk of these exposures is low, very large numbers of exposed persons are required for study if the outcome is to have any decent statistical power.

## PROBLEMS IN CURRENT STUDY DESIGNS

From the previous discussions, four areas of major problems become evident: 1) the assessment of the exposure-response sequence; 2) quantification of exposure; 3) recognition of bias and confounding; and 4) quality and validity of data. Clearly, a very complex study design may be required to yield useful results.

Measures to improve the usefulness of human studies for risk assessment purposes include the extension of the duration of follow-up time, assessing the time component in exposure and disease diagnosis, focusing on potentially high-risk populations for study, and quality assurance of information on exposure and disease. While most of these measures are in the area of logistics and funding, an important exception is improvement of the quality of the exposure data.

In the past decade, development of environmental exposure mesures has been very rapid. Detection limits for chemicals in environmental media have dropped by three to four orders of magnitude, and the progress of tests for some chemicals in biological media is almost as impressive. The detection limits for dioxin in sera, for example, is now measured in parts per quadrillion. Unfortunately, little progress to date has been found to be of practical use in epidemiologic analysis and risk estimation. For instance, issues of background levels, biological persistance, adaption mechanisms, absorption kinectics, saturation of metabolic pathways, and the impact of an individual's characteristics on the pathogenetic process have not been addressed in most epidemiologic study designs, and, for the most part, have yet to enter the area of regulatory risk assessment.

There are other practical exposure issues that need to be addressed such as noncontinuous or fluctuating exposures, the cause of interspecies differences, and whether or not an observed dose-response relationhip is stable over a wide range of dose levels. We will also see an increasing demand to incorporate quality assurance and quality control in epidemiologic studies with regard to matters other than laboratory work. For example, it is of utmost importance to make certain that the disease of concern is following and not pre-dating exposure. Finally, there is the issue of the quality of the diagnostic criteria for a case or a non-case.

The quality of diagnosis becomes a very central issue when it comes to scenarios of localized environmental pollution, for example, at a chemical dump site, and residents with nonverifiable and subjective complaints, which may be real to them, sucah as headache, fatigue, nausea, chest pain, and loss of libido. Currently, there is an inclination among epidemiologists to ignore or disqualify this so-called "dump-site syndrome" from serious study. However, such an attitude is usually followed by a deterioration of a conflict situation between citizens and authorities. There are many instances where eventually epidemiologists have been forced by heavy and relentless public and political pressures to conduct studies of such perceived illnesses. In doing so, they will have to derive methods to cope with non-verifiable health outcomes, while maintaining scientific integrity and credibility. In theory, it should be possible to either solve the problems with statistical tools, or by developing tests for the kinds of complaints often described as emotional or behaviorial. CDC staff are currently developing and applying such tools to several large studies.

Statistical methods usually fail since the situation at a dump site is inherently associated with an abundance of negative publicity, usually in the direction of stating the association of voiced complaints with exposure, or even just living near a dump site, as a fact. This scenario often results in serious reponse biases for persons who perceive they may be exposed. I do not forsee that behavioral toxicology, an exciting new field of research, can provide us in the near future with the appropriate scientific tools to address currently nonconfirmable complaints.

## DEVELOPMENT OF MOLECULAR EPIDEMIOLOGY

A special problem, both in animal and human studies, is that current designs deal with observed disease, which is a more or less advanced stage of a toxic effect. In animal studies, most diseases are observed in moribund or sacrificed animals. In humans, disease detection is usually in an earlier phase by virtue of man's ability for detailed communication. However, even common diseases such as cancer, arthritis, hypertension, and diabetes still pose unresolved problems in assessing the date of onset. Estimates of this date may differ by many years, and this would offset greater accuracy in exposurement. The logical response to this problem is to develop techniques to diagnose the disease in the earliest possible stage. But the question then arises: "What is earliest possible?" An aggressive biopsy regimen for diseases such as cancer and kidney disease may shift the date of diagnosis from months to years earlier. Certain inborn metabolic disorders can now be detected prenatally. The use of electron microscopy has brought us closer to the early onset of renal disease. Unfortunately, these striking improvements in early diagnosis require invasive procedures. This is a serious handicap to epidemiologic studies, especially those involving environmental rather than clinical or occupational exposures. This explains the increasing interest of epidemiologists and risk assessors in the use of biomarkers indicating past exposures or early stages of tissue dysfunction, for example, DNA-adducts.

## VALADIATION OF ASSUMPTIONS FROM EPIDEMIOLOGIC STUDIES FOR REGULATORY PURPOSES AND PUBLIC HEALTH POLICY DECISIONS

Finally, I would like to discuss the interest of epidemiologists in the validation of a number of assumptions used in risk assessments for regulatory purposes or public health policy decisions. One of these is the assumption that the presence of a toxic chemical in the environment automatically implies exposure, and that that body dose is proportional to environmental concentrations. This assumption leads to the often-used, but nevertheless incorrect practice of assuming that the concentration of a chemical in media such as soil, air or water is a direct measure of the amount of chemicals absorbed in the human body. Worse, without much thought it is often considered identical to the challange to the organ or tissue interest when determining acceptable exposure levels. Studies into the relation between environmental presence, human exposure, and organ-specific dose are increasing in number.

The findings from these studies have sometimes been contrary to expectation. For example, at the CDC, studies have shown that the concentration of arsenic, PCB's, mercury, and lead in the soil of a neighborhood is only partly related if at all to the levels in the biologic specimens of residents. In this light, it is important to recognize the importance of well-conducted research with negative findings. Such research is critical to our understanding of the effects of toxicants on human biology. Moreover, such findings help concerned scientists to inform the public of true risks and allay undue anxiety. Indeed, despite the abundance of available data to date, the relation between environmental concentrations of chlorinated hydrocarbons such as DDT, dioxin, and PCB's, and human sera or adipose samples, remains unclear, and the relation of these levels of body burdens to clinical disease remains uncertain.

To date, epidemiologic studies almost never prove cause and effect, though in a few instances, reasonable people would accept some of them as such. For example, in looking at the pathway of exposure and body burden, the association of the reduction of lead used in gasoline production and the reduction of mean blood lead levels in the U. S. population is striking. Over a 4-year period when the lead phasedown in gasoline was occurring, we were conducting a study of blood lead levels in the U. S. population using data from the Second National Health and Nutrition Examination Survey or NHANES-2, an example of a cross-sectional study. Two things, declining blood lead levels and lead used in gasoline production were highly correlated. We removed over 100 potentially confounding variables from this association in the analysis and the coefficient of correlation did not appreciably change. Yet many epidemiologists stated that this did not provide adequate evidence of cause and effect. The only way to unequivocally prove cause and effect in this situation would be to conduct an experimental study where children were placed in chambers and breathed air with different lead levels and then measure their blood lead levels. This experiment, of course, would be entirely unethical and would not be supported by society. Studies conducted in humans must use only inadvertant exposure or "natural experiments" such as that occurred with water fluoridation and dental carries.

Proper use of epidemiologic data can lead to important collective public health benefits. On the other hand, to press such data into service to respond to causal effects for an individual's disease holds high potential for misuse of the data.

We will continue to respond to specific incidents of human exposure to toxic or hazardous substance . We will also continue our efforts, through epidemiologic techniques, to measure both the immediate and long-term health effects and to make sound recommendations for the attentuation of these potential risks.

Although the results of such epidemiologic investigations may not provide the conclusive answers about health risks from environmental exposures, which are now in such demand and so prevalent in the media, we have hope that we can study and detect these associations where they exist, so that prudent public health actions can be taken. Thus, we see the ultimate role of epidemiology as one of prevention, which is the most effective public health policy to implement.

# SUGGESTED READINGS

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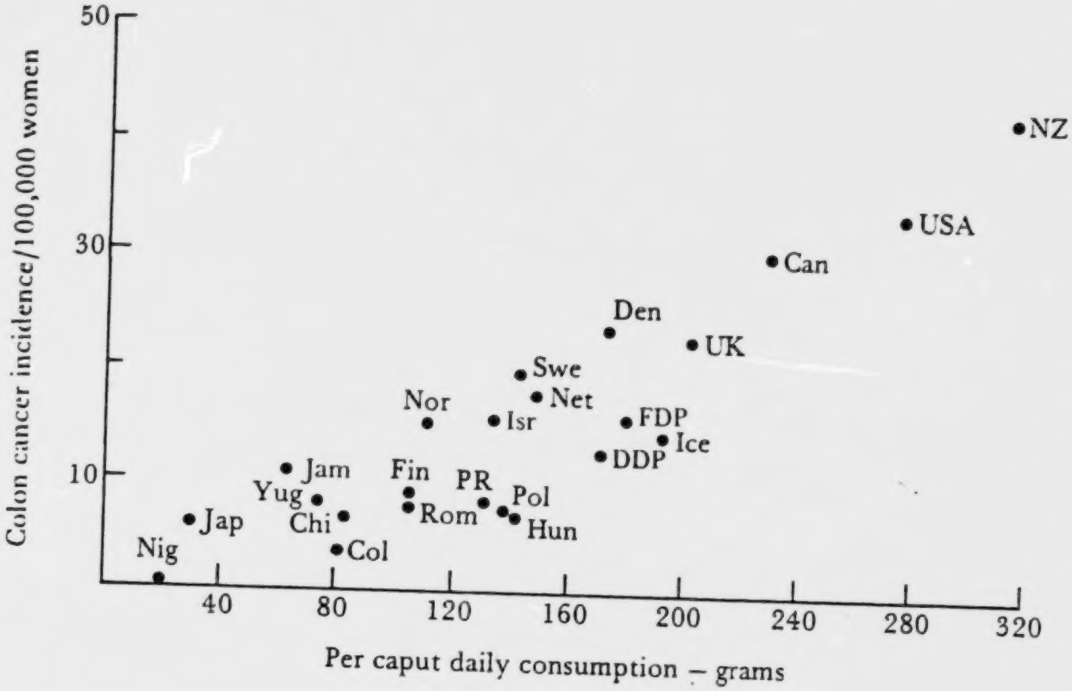

Figure 1. Colon Cancer Incidence Rates/100,000 by Per-Capita Meat Consumption (grams) for Selected Countries

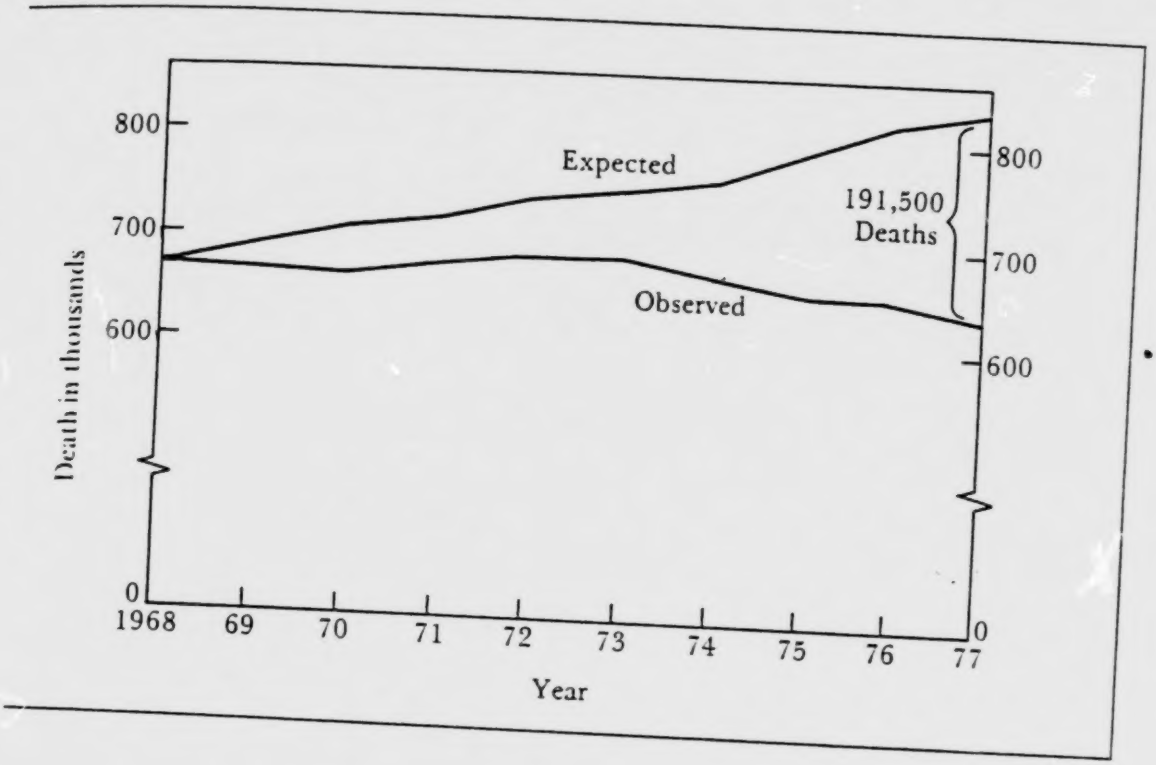

Figure 2. Difference Between Observed and Projected Mortality Cases from Coronary Heart Disease, United States (If deaths in 1977 occurred at the same rates as in 1968)

**DRAFT**

AERE Conference

June 8, 1989

# Malignant Melanoma Death Rates,
# Outdoor Recreation
# and
# Sun Screens

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Economic Studies Branch
Office of Policy Analysis
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May 18, 1989

Disclaimer: This paper reports interim results on research which is still in process. It presents the personal opinions of the author. It has not been reviewed by EPA and does not represent an official EPA position.

# Abstract

Previous work has shown that there is a six fold increase in the risk of death from melanoma for white males born in the 1940's when compared to white males born in the 1880's and 1890's. For women the same ratio is slightly less than three. Accepting the hypothesis that most melanoma is caused by exposure to solar radiation, an investigation of changes in residence patterns, occupation, and outdoor recreation is made to see if the changes in cohort risk can be explained by changes in factors related to exposure patterns. Household access to automobiles turns out to be the best potential measureable factor explaining outdoor recreation patterns. While no conclusive findings are reached, support is developed for the hypothesis that intense exposure of skin which has not developed natural defenses under low to moderate exposure is the primary risk factor for melanoma. The introduction of sun screens is associated with reductions in this risk. Since lifetime incidence rates for white males in the 1940's cohorts will approach 2.5 percent with death rates of about .6 percent, melanoma is a significant public health problem. The paper's results suggest that a risk communication policy should be aimed at modifying sun exposure habits to reduce intensity of exposure. The association of the automobile with problem exposure behavior suggests a strategy of keeping sun screen in the glove compartment. The payoff from such a policy could be a dramatic reduction in melanoma incidence and death.

## INTRODUCTION

Cutaneous malignant melanoma is the most rapidly rising cause of cancer death for white males and the second only to lung cancer for white females.[1] In response to this, there has been much investigation of potential causes.[2] Because melanoma cannot be induced in small laboratory animals by ultraviolet radiation alone, the ability of laboratory research to settle etiologic issues has been sharply limited.[3] Epidemiologic results have been inconsistent, with less melanoma observed on frequently exposed parts of the body, and death rates increasing with latitude in Europe.[4,5] Occupations involving outdoor exposure have been found to be mildly protective.[6] It has been difficult to develop a model which can comfortably explain all of these results. Thus, despite all the work on melanoma to date, there is still a clearly understood feeling by the research community that this is a disease whose etiology is not at all well understood.[8]

The first part of the paper reviews some basic biological and epidemiological results. The next section reports on previous results obtained in this research project. The project has been centered around factors affecting melanoma death rates for US whites between 1950 and 1984. County death rates have been agregated into Standard Metropolitan Areas(1980 definition) and merged with census data on sociodemographic characteristics of the 1980 population, weather data for each city, and model based estimates of exposure.[9] This data set has been used to investigate the response of death rates to potential exposure, the cohort structure of death rates, and the response of death rates to individual components of the ultraviolet spectrum. For this paper, the data set is used to predict cohort levels of risk, which serves as the basis for the analysis of changes in ecologic risk factors.

The third part of the paper then procedes to examine how factors such as outdoor recreation, outdoor work, and residence have varied over the period for which cohort risk of death of melanoma can be inferred from the data set. First, some measures of how these factors have changed are developed. These are then compared to the summary risk measures for each cohort. Out of this there emerges a fairly clear picture of the kinds of exposure factors that can be related to the observed change in risk. These factors can explain the rise and stabilization of the cohort risk factors. They cannot explain the downturn in risk seen in the youngest cohorts.

The next section looks at available data on sun screens to see if they are a potential cause of the downturn. It is shown that sunscreens can be an explanation of the decline only under the hypothesis that it is control of intense exposure of skin which has not developed natural protection which is important if risk is to be reduced. Usage levels are too low for them to have been a factor if control of all exposure is necessary to reduce risk. The last section developes some of the potential benefits of a risk communication strategy developed along the lines suggested by the results of the previous sections.

## BACKGROUND

This section reviews some basic biology, some of the little that is known about how the skin develops natural protection, and some results about how exposure changes as a function of latitude, time of year, and time of day.

Melanoma arises in the melanocyte, the cell which produces melanin, the compound responsible for skin color.[10] The precise process by which the transformation to a tumor takes place is not known.[11] The tumor is normally highly antigenic--meaning the immune system will attack it--and one of the clinical markers for an early lesion is a red iritated area around the lesion.[12] Since UV radiation is known to suppress some aspects of the immune sytem, immune suppression via this route is thought to play some role in the disease.[13] However, this role remains to be worked out in detail. The tumor metastizes readily once it penetrates the surface of the skin and it is the metastases which are responsible for the mortality associated with melanoma.[14] On the other hand, five year survival rates for melanomas removed before the dermis has been invaded are about 95 percent.[15] Thus early diagnosis and removal are critical to effective treatment of the disease.

Incidence and death rates from melanoma have been growing very rapidly.[16] Figure 1 shows death and incidence rates for whites in the US. In 1984 total deaths from melanoma in the US were 5377. Of these deaths 5264 were whites and 113 were non whites. Age adjusted death rates were 3.11 for white males, 1.65 for white females, .37 for non-white males and .41 for non-white females. This is a world wide pattern, indicating that melanoma is primarily a disease of white populations. For non-whites, melanoma almost always arises in the non-pigmented portion of the body, either under the nails or on the soles of the feet.[17] Thus pigmentation is protective. This is true even within the white population, with southern Europeans such as Spanish and Portuguese much less likely to get melanoma than those of northern European origin.[18]

The hypotheses that melanoma might be solar related stems from the fact that non-melanoma skin cancers seem to be clearly sun related.[19] Non-melanoma cancers occur most frequently on the exposed portion of the body, and are much more frequent on those with lots of outdoor activity--thus they clearly are a function of lifetime exposure.[20] Melanoma, on the other hand does not follow this pattern.[21] Less exposed parts of the body, such as the trunk in males, and the legs in females, are the predominate place where melanoma is found. This clearly indicates the need for some modification of the solar hypothesis. The second problem stems from the results for Europe, which show that the expected decrease of melanoma incidence and death rates with latitude does not occur.[22] Rather rates are lower in southern Europe than in northern Europe. This may be due to the pigmentation variations disucussed earlier. Later results in the paper on the possible role of recreation, occupation, and residence patterns may also help explain the anomaly.

Existing results also point to the role of early exposure as being critical.[23,24] Again, the results are not unambiguous, and in some cases depend on quite small samples. Finally, due to the lack of an animal model, the exact portion of the spectrum responsible for carcinogenisis is not clear.[25] The hypothesis is that the UVB part of the spectrum is responsible, since this is the part of the spectrum where damage to DNA occurs. Due the lack of a widely distributed network of instrumentation capable of individual waveband measurement, there has been no confirmation of this by epidemiological studies. Thus the potential role of sunscreens as a protective device has been difficult to determine since the major chemicals are effective only in the UVB part of the spectrum.[26]

Exposure to the sun elicits the production of melanin and the development of a thicker stratum corneum, the outermost layer of cells on the skin.[27] Both of these factors reduce penetration of UV radiation to the growing layer of cells. While it is difficult to determine the exact extent of the protection induced by these factors, the tanning process does increase the length of time necessary to produce erythema(sunburn) by at least a factor of three.[28] Black skin reduces the level of radiation reaching the melanocyte by about a factor of 10.[29] Perhaps obviously, the incident angle of radiation is also very important, since radiation entering the skin at a sharp angle must travel much further before reaching the growing layer of cells. Thus, most work activities expose substantially less of the skin to intense doses than do activities like sunbathing, where the body is prone.

Ultraviolet radiation present at ground level starts at about 290nm and increases by about 5 orders of magnitude in intensity by 325 nm. From this wavelength to 400 nm, the lower end of visible spectrum, radiation is rougly constant in intensity and varies in the same manner as visible light. The large variation in intensity between 290nm and 325nm is due to absorption by ozone in the stratosphere. Figure 2 shows variation in DNA weighted radiation by latitude for a clear day in the peak month of the year and for total radiation during the year. Note that there is little variation in peak values between the equator and 30 degrees latitude. Figure 3 shows DNA and Erythema weighted radiation measures during the year for Washington DC. Note that DNA weighted radiation varies more than does erythema weighted radiation. Figure 4 shows variation during a peak day in July. Note again that DNA radiation varies more during the day than does erythema. The relevance of these differences in behavior will become clear later in the paper.

## PREVIOUS RESULTS FROM THIS PROJECT

The work already done on this data set bears on a number of the open questions discussed above. First, it shows that variations in intensity of ultraviolet radiation are associated with higher death rates.[30] A one percent increase in peak(clear summer day) DNA weighted radiation yields a .85 percent increase in the death rate for males and a .58 percent increase in the death rate for females. Controls for socioeconomic variables do not affect the results while including the effect of ethnic origin reduces the responsiveness of death rates by about 20%.

The second area of work with this data set suggests that it is exposure in the UVB part of the spectrum which is responsible for the carcinogenisis.[31] The exposure measures used in previous epidemiology on melanoma have been simple latitude(which is a non-linear function of exposure as figure 2 illustrates), hours of sunlight, or an integrating meter(known as the Robertson Berger meter) which gives a single measure of UV radiation.[32] The exposure measure used in this study is developed from a model which incorporates satilite measures of ozone into a radiative transfer model to predict ground level UV radiation. These predictions can either be in the form of wavelength weighted measures where the weights are the inverse of the biological effectiveness of different wavelengths, or as individual waveband energies. In this particular work, individual waveband energies from 295-299 through 330-334 for a clear day in June were used as exposure measures. Table 1 presents the estimates for different wavebands. Deaths were modeled as a poisson process and estimation was done using iteratively reweighted least squares to get maximum liklyhood estimates. The results show a positive relationship between radiation below 320, with a negative and significant relationship above 330 for males. For females that pattern is similar, but the results are not significant above 330nm. Because of high correlations between different wavebands, it is not possible to introduce more than two wavebands simultaneously intc the equations. The second part of Table 1 shows the results using 295-299 and 300-304 as the short waveband with various wavebands used as the long waveband. This indicates the upper range for positive response to radiation lies at about 315 nm or at the upper end of the range where radiation damages DNA.

These results rely heavily on variations in specific parts of the spectrum. Since the model has only been tested with aggregate mesures produced by the Robertson-Berger meter, more work is needed to baseline the model. However, the overall pattern of variation is dependent only on variations in measured ozone and very basic radiative principles. Thus while there may be measurement error, it is unlikely to be systematic in nature, and thus, in this simple model, the expected result would be to bias the estimated coefficients toward zero.

The third set of analyses done with this data look at cohort experiences.[33] As seen in Figure 5, there is a very systamatic structure to a plot of the log of the national cohort death rates against age. Cohorts are defined as those who are 0 to 4 years of age for a five calender year interval. This results in a median birth year equal to the initial calender year of the period. This definition was required because death data were only available in five year age groups in the source data set. The labels on the plot refer to the median birth year for each cohort. The parallel slopes of the cohort death rate curves above the age of thirty suggest that it is early exposure which is critical to the potential risk. Statistical analysis confirms that the curves above the age of 30 have equal slope. For men this slope is 7 percent per year and for women it is five percent per year. As Figure 5 shows, there is no slope to the death rate experience before age 10. Clinical experience indicates these deaths are due to congenital nevi.[34] Therefore, it seems reasonable to assume that the rate for 0 to 9 year olds is constant, and all the variation in cohort risk is due to variation in how the death rate changes between age 10 and age 30. Table 2 presents estimates for a model which includes DNA weighted exposure, individual cohort estimates for $7 < Age < 32$, and a common age effect above age 32. These results suggest that variations in some aspect of exposure across time for the age group less than 30 are at the root of the varying coefficients for the cohort specific age variable.

Using a much simpler procedure, estimates at age specific rates at age 32 can be made for chorts born between 1865 and 1970. For the 35 years of data available, average ratios for each five year differential are computed. These averages are used to extrapolate from the nearest available death rate to the age 32 death rate. Table 3 gives the results of these forecasts for each birth cohort. White males show marginally greater than a ten to one variation while white females show about a five to one variation. Interestingly, there is a predicted downturn in the age specific rates for cohorts born after 1950. As seen in figures 5 and 6, these reductions are already seen in these cohorts at younger ages.[35] Next to the differential rates for blacks and whites, this is the largest variation seen in experience with melanoma. Thus any explanation of melanoma aetiology must deal with this experience. The next section of the paper looks at some potential explanations for these large cohort effects.

## COHORT VARIATIONS IN DEATH RATES

Given the small number of degrees of freedom across cohorts and the very limited quantity and quality of data on recreation in particular, the analysis in this section is more qualitative in nature that the analysis in the previous sections. The essential question to be addressed is what changes have occurred in exposure habits and opportunities between 1880-84, when the oldest cohort in the study was 15-19 years old and the 1980-84 period, when the 1965 cohort was 15 to 19 years of age. There are a number of hypothesis which could be suggested for the variation across this period of time. Here only solar related hypotheses are considered since there is little indication in the literature of any other cofactor besides genetic predisposition as a potential cause of melanoma.(This is not to say one might not exist--but only that a creditable one has not been found so far).

The first potential hypothesis is that changes in place of residence during the critical exposure years might have changed so that average intensity of exposure is higher. However, as Table 4 shows, DNA relevant radiation weighted by state populations between 15 and 24 for every five years between 1890 and 1985 increases by only 2.8 percent in intensity(average exposure in 1980 is 3.25). Since this would amount to only an 2.5 percent change in risk at age 32 for males and a 1.65 percent change in risk at age 32 for females, this does not explain the very large changes in lifetime risk seen in Table 4.

Likewise, occupational exposure is not the explanation. The two major occupation groups with extensive sun exposure are farming and construction. As Table 5 shows, these have fallen sharply in relative size, and even in absolute size during the 1880-1985 period. Also occupation is less apt to be a risk factor for those under the age of 20 since labor force participation rates are relatively low and have been quite static in the 50 to 60 percent range for white males between 14 and 19 and between 20 and 30 percent for white females in the same age group.

A third potential hypothesis centers around outdoor recreational exposure. This can at best be a partial explanation of the changes in melanoma risk. Around 1900, forty percent of the population lived on farms and participation in outdoor recreation was about 4 hours per capita per year(see Table 7), while the lifetime risk of death from melanoma was only about 1 in 1000 for both males and females. In 1960, eight percent of the population lived on farms, per capita participation in outdoor recreation had risen to almost 120 hours, and the lifetime risk of death from melanoma had reached 6 per thousand for males and 2.7 per thousand for females. From 1960 to 1985, farm population fell to about 2.5 percent of total population, per capital outdoor recreation hours by 2 and one/half fold, but the risk of melanoma has decreased. While the results between 1900 and 1960 are suggestive of a role for outdoor recreation, the 1960 to 1985 results suggest(as always with melanoma it seems) that if there is a role for recreational exposure it is not a simple one.

One can make sense of the role of recreation if what matters is not the extent of participation, but simply participating at all. Under this hypothesis one would expect to see a stabalization in participation rates in sun intensive activities beginning in the sixties. Unfortunately, data on a comparative basis does not exist. What can be examined is a number of proxy variables for participation. One proxy for recreation behavior is the percent of the labor force not at work due to vacation(see Table 8). While comparable data is not available before World War 2, data given in Clawson and Knetch indicate weeks of vacation per worker rose from .37 in 1929 to 1.09 in 1959, suggesting percent participation rose during the 1929-1946 interval also.

A second indicator of percent participation comes from noting that over ninety percent of outdoor recreation involves automotive transportation.[xx] Assuming that most recreational activity involving automotive transportation is family oriented, the critical variable controlling access to outdoor recreation is household motor vehicle ownership. Table 9 gives this data for the post war period. Table 10 extends this back before WW II as mean vehicles per household. Comparison with Table 9 indicates that mean vehicles oer household is roughly double the percentage of households owning at least one car. What is clearly interesting about this variable is the apparent saturation on a per household basis which occurs in the early 1960's.

These two variables suggest that a case can be made that the breadth of participation in outdoor activity stablized in the 1960's. Several surveys of participation in outdoor activities were done between 1962 and 1982-83. While summary results from these surveys are not in a format that makes comparison across time possible, the latest survey(1982-83) does indicate that all but 11 percent of the general population participate in some form of outdoor recreation. For those between 12 and 24, all but 3 percent of the population participate. The next step in this process is to get the source level documents and see if a more coherent picture can be developed.

The other aspect of changes in recreational exposure which is important to understand is that activities associated with intense exposure have increased over time. Swimming, especially sunbathing, is typically associated with more intense exposure than hiking or bicycling. Further, in the northern part of the country, the outdoor swimming season does not begin until the intensity level is within ten percent of the peak level it will achieve during the year. One illustration of the increase in intensity is that the number of muncipal swimming facilities per capita increased more than seven fold between 1910 and 1965.

The issue of when expsoure begins, alluded to in the previous paragraph, is also important. There have been very significant changes in the time at which exosure begins for the critical age groups. In the 1880's, they typical student attended school for only 80 days a year, and stoped school after the eighth grade. Today, the typical student attends school twice as long and in excess of 95 percent of the 5-17 year old population is in school. Given the shortness of the typical school year before 1900, we can suppose that the real pattern for farm children, especially those in the early teens, was to be outside helping with farm work beginning in the spring and continuing through the fall. This is not a pattern of limited sun exposure. It is a pattern which leads to the development of a tan prior to the period of peak intensity. As Table 1b shows, if the participation in farm work begins in March or April, the exposures levels are much lower than those found beginning in late May or June, the typical time at which school closes in the modern era and outdoor recreation starts.

Thus we have a potential hypothesis explaining the increase in melanoma as a function increase in the effective intensity at the time when sun exposure begins for the season. Under this hypoothesis, the stabilization of rates occurring in the 1930's to 1950 for females and in the 1950's for males is explained by stabilization in the percent of the population getting intense exposure. The large increase in recreation behavior since the sixties is one of more extensive participation by each individual, rather than a broader participation. If this is in fact the case, and more work is needed on this score, then a consistent pattern can be found which explains the growth of melanoma by an increase in the effective intensity of radiation brought about by changes in education and work patterns which delay the onset of outdoor exposure until the period of peak insolation, and the spread of activities such as swimming, which expose much of the body to sunlight, especially parts of the body which rarely receive any prior exposure.

This would explain the markedly lower rates seen for the head neck and hands for melanoma, since these parts of the body are exposed year round and thus have always developed some level of natural protection. It does not however, explain the decrease in melanoma rates seen in cohorts born since the 1950's. One possible explanation, consistent with the solar hypothesis, is that sun screens have played a role. This is discussed in the next section.

**SUNSCREENS**

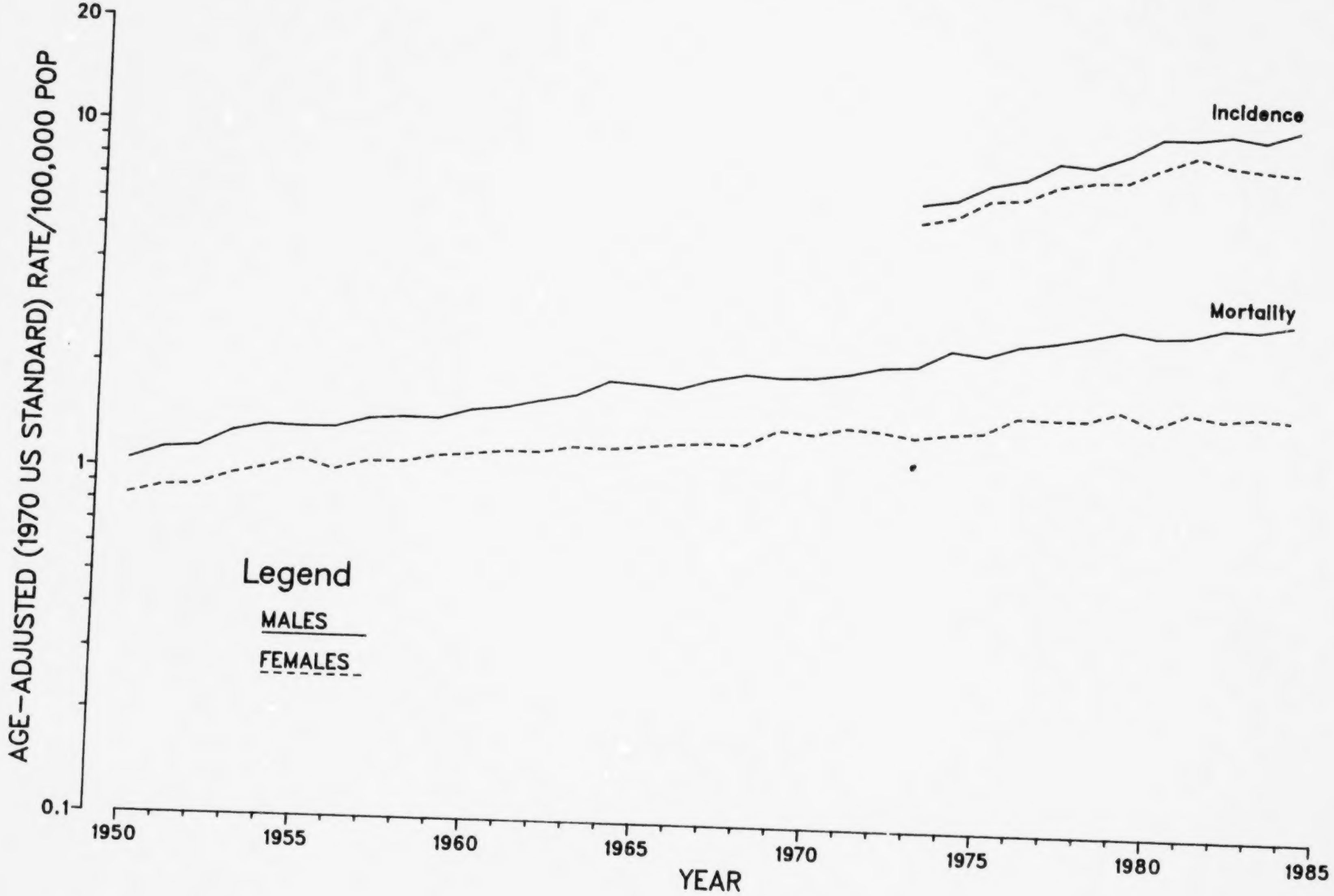
Under the intensity hypothesis, to be effective in reducing risk, sunscreens do not have to be used all the time, but only during the period of initial exposure. The question is whether the total use of sunscreens, given in Table 12, is sufficiently high to have possible been effective in reducing risk. To provide adequate protection at the rated level, about one ounce of product is required.[xx] Typical applications to the entire body seem to be at about half this rate.[xx] This rate is still enough to produce a very significant reduction in risk. Thus the actual number of applications available is about twice the number of ounces sold. This yields about three applications per individual, which is probably a minimum level of protection for one day on the beach, but not enough to get all potentially exposed individual through the period of developing a tan without getting an intense dose. Thus it is unlikely that sun screens are the potential explanation for the declining risk seen in the younger age cohorts. Only if all sunscreen use were concentrated in the younger cohorts would this be possible. In fact, anecdotal evidence suggests it is the younger cohorts which are least apt to use sunscreens. Thus some other explanation for the decline must be sought. This does not mean that a policy of increased use of sun screens would be ineffective. The potential of such a policy is discussed in the next section.

## FOUNDATION OF A RISK ASSESSMENT POLICY

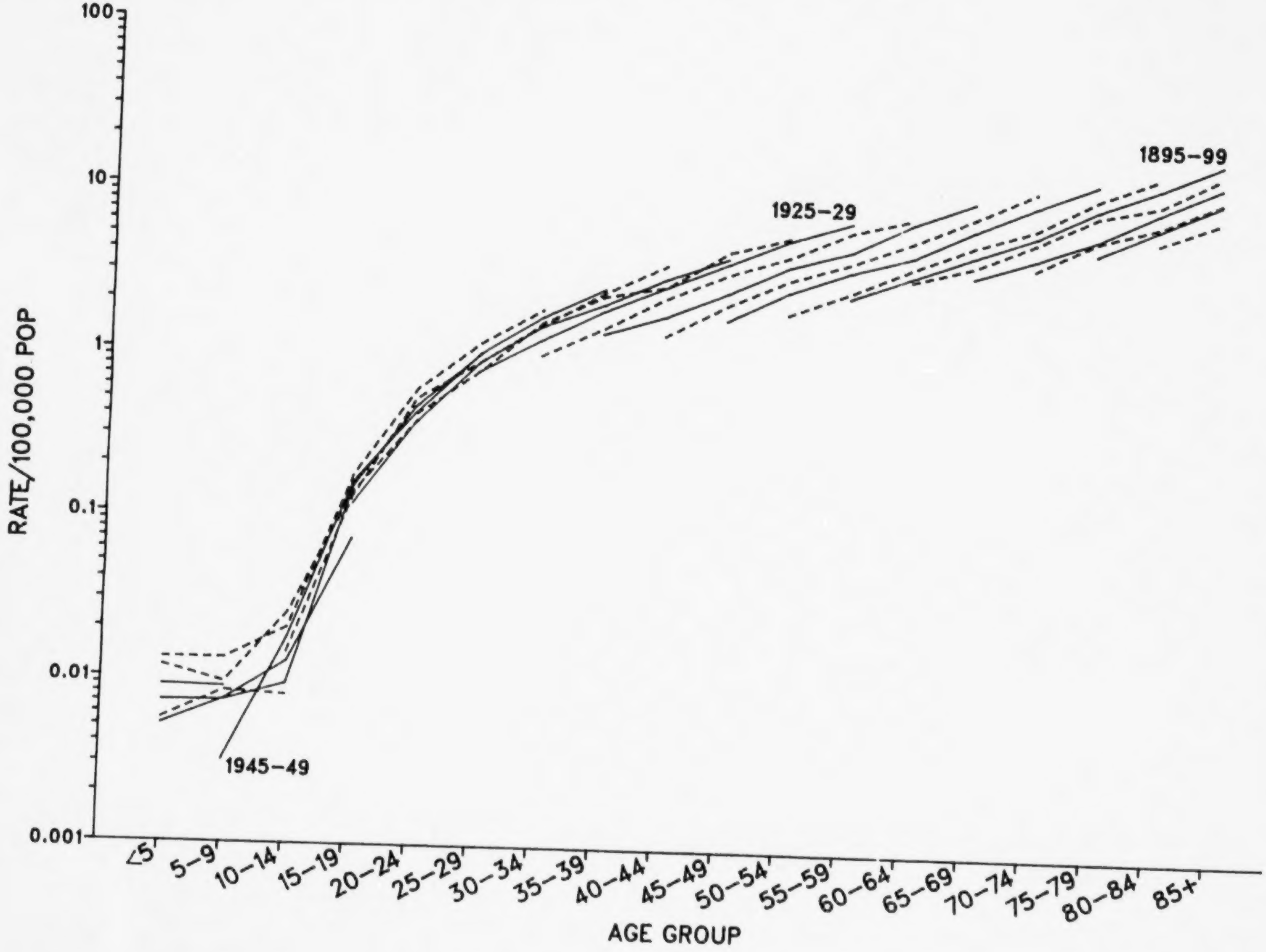
The intensity hypothesis suggests that a policy is possible which might be very effective in reducing melanoma. The primary goal of the policy would be to limit exposure very carefully during the period before the skin has a chance to develop its natural defenses of thickenign of the stratum corneum and tanning. Since these processes both take time, this would imply either the careful use of sunscreens or a significant limitation on activity during a vacation taken by somebody who starts exposure when natural intensity levels are high or travels to a sunny area during the winter.

How much might such a prevention program be worth. No strategy could probably return us to the results of the 1900 era. There is simply too much intense radiation present in modern recreational activities and use of sun screens is unlikely to be universal. However, it might be reasonable to reduce risk by 60 percent. The results of a policy with this level of effectiveness are illustrated in Table 12. Since we have a fairly detailed sense of the death rate pattern for melanoma, the table looks at years of life saved rather than reductions in mortality. The figures are for a cohort group of 100,000. The total reduction in melanoma mortality, in a given year, under steady state cohort behavior, would be 368 lives per 100,000 males and 162 lives per hundred thousand females. At currently typical white birth cohort sizes of about 1.75 million each for males and females, this yields a total reduction of better than 9000 melanoma related deaths. Total associated incidence would be about four times these levels, giving reduced incidence of about 36,000 cases. It should be emphasized that these are long run numbers and do not take account of whatever is currently acting to reduce death rates. They do suggest that a policy to moderate sun exposure habits has a very high potential public health payoff.

# Skin Melanoma Trends Among Whites in the United States



# Skin Melanoma Mortality by Birth Cohort Among WHITE MALES



## Skin Melanoma Mortality by Birth Cohort
## Among WHITE FEMALES

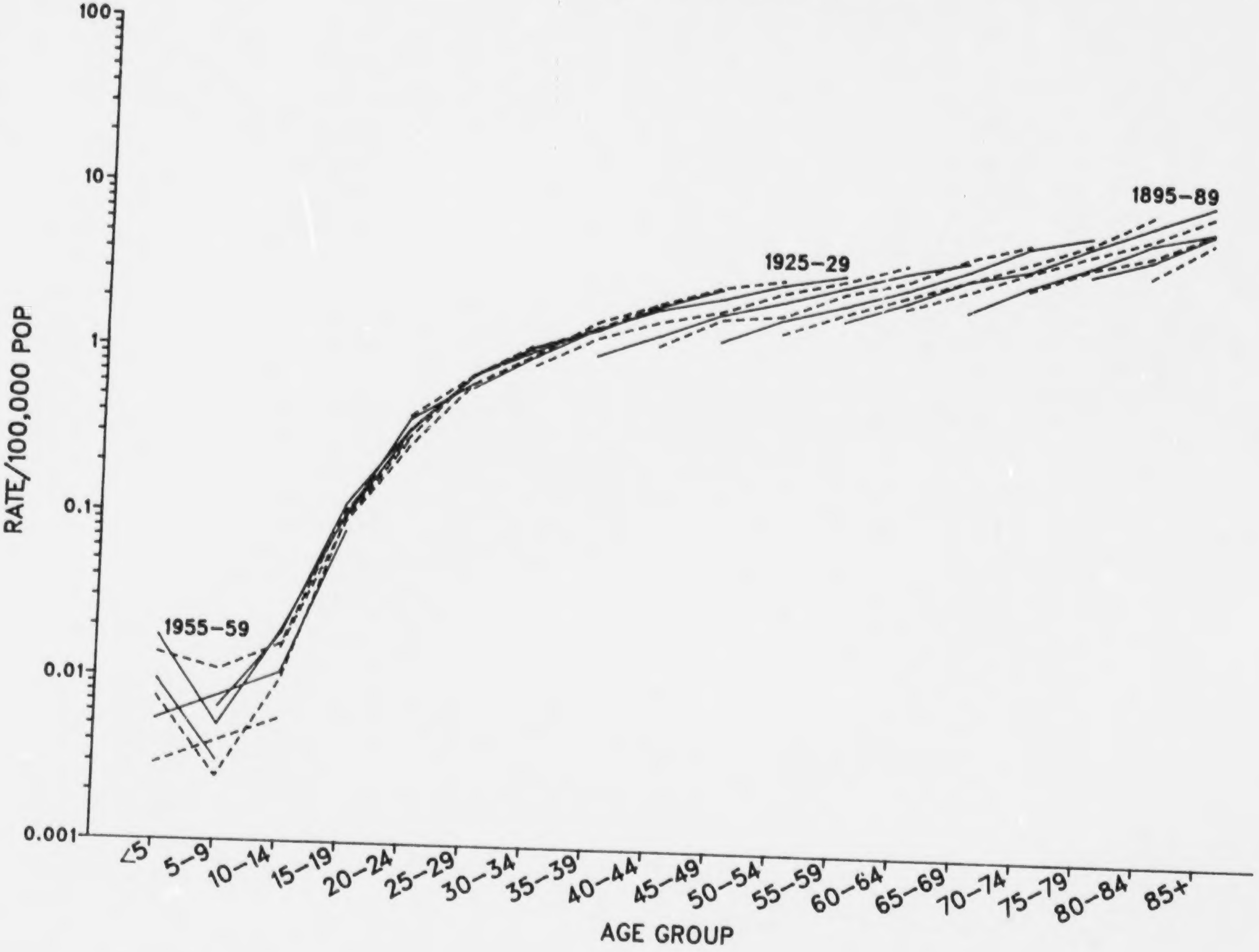


Table 1a

Ultraviolet Radiation Variation by Latitude

| Latitude | Clear Day DNA Weighted | Annual DNA Weighted |
|---|---|---|
| 0 | 5.45 | 1162.5 |
| 10 | 5.39 | 1181.6 |
| 20 | 4.59 | 989.1 |
| 30 | 4.14 | 658.0 |
| 40 | 3.31 | 370.7 |
| 50 | 2.33 | 204.0 |
| 60 | 1.67 | 124.3 |

Table 1b

Clear Day UV Radiation by Month

| Month | DNA Weighted | Erythema Weighted |
|---|---|---|
| Jan 15 | .25 | 31.0 |
| Feb 15 | .50 | 57.9 |
| Mar 15 | 1.01 | 108.7 |
| Apr 15 | 1.85 | 186.8 |
| May 15 | 2.51 | 246.5 |
| Jun 15 | 3.14 | 299.6 |
| Jul 15 | 3.40 | 318.3 |
| Aug 15 | 2.91 | 274.6 |
| Sep 15 | 2.03 | 196.2 |
| Oct 15 | 1.09 | 110.9 |
| Nov 15 | .46 | 51.2 |
| Dec 15 | .24 | 28.9 |

Source: Model based estimates using satelite data on ozone. Units are not comparable.

## Table 1c

UV Radiation on July 15(Clear Day)

| Time of Day | DNA Weighted | Erythema Weighted |
|---|---|---|
| 6-6:30 am | .0055 | .78 |
| 6:30-7 am | .0129 | 1.66 |
| 7-7:30 am | .0259 | 3.06 |
| 7:30-8 am | .0456 | 5.03 |
| 8-8:30 am | .0723 | 7.55 |
| 8:30-9 am | .1050 | 10.53 |
| 9-9:30 am | .1419 | 13.77 |
| 9:30-10 am | .1800 | 17.05 |
| 10-10:30 am | .2162 | 20.10 |
| 10:30-11 am | .2469 | 22.66 |
| 11-11:30 am | .2693 | 24.51 |
| 11:30-12 am | .2811 | 25.48 |
| 12-12:30 pm | .2811 | 25.48 |
| 12:30-1 pm | .2693 | 24.51 |
| 1-1:30 pm | .2469 | 22.66 |
| 1:30-2 pm | .2162 | 20.10 |
| 2-2:30 pm | .1800 | 17.05 |
| 2:30-3 pm | .1419 | 13.77 |
| 3-3:30 pm | .1050 | 10.53 |
| 3:30-4 pm | .0723 | 7.55 |
| 4-4:30 pm | .0456 | 5.03 |
| 4:30-5 pm | .0259 | 3.06 |
| 5-5:30 pm | .0129 | 1.66 |
| 5:30-6 pm | .0055 | .78 |
| 6-6:30 pm | .0020 | .30 |
| 6:30-7 pm | .0006 | .09 |

All radiation values from radiative transfer model incorporating satelite measurements of ozone.

## Table 2a

## Wavelength Specific Estimates of Exposure Effects*

### Males

| Wavelength | Coefficient | Standard Error |
|---|---|---|
| 295-299nm | .142 | .0104 |
| 300-304nm | .148 | .0108 |
| 305-309nm | .153 | .0115 |
| 310-314nm | .145 | .0125 |
| 315-319nm | .112 | .0132 |
| 320-324nm | .055 | .0139 |
| 325-329nm | .0014 | .0143 |
| 330-334nm | -.028 | .0143 |
| 335-339nm | -.044 | .0136 |
| 355-359nm | -.064 | .0138 |

### Females

| Wavelength | Coefficient | Standard Error |
|---|---|---|
| 295-299nm | .0817 | .0123 |
| 300-304nm | .0871 | .0128 |
| 305-309nm | .0923 | .0135 |
| 310-314nm | .0912 | .0144 |
| 315-319nm | .0765 | .0152 |
| 320-324nm | .0458 | .0157 |
| 325-329nm | .0159 | .0158 |
| 330-334nm | -.00090 | .0158 |
| 335-339nm | -.0106 | .0149 |
| 355-359nm | -.0204 | .0152 |

## Table 2b

### Multiple Waveband

| | Male | Female |
|---|---|---|
| 295-299nm | .151<br>(.0182) | .0709<br>(.0219) |
| 315-319nm | -.0255<br>(.0197) | .0197<br>(.0225) |
| 295-299nm | .150<br>(.0114) | .0804<br>(.0136) |
| 320-324nm | -.0298<br>(.0152) | .0012<br>(.0176) |
| 300-304nm | .166<br>(.0124) | .0908<br>(.0147) |
| 320-324nm | -.0472<br>(.0158) | -.0091<br>(.0182) |

* All units have been converted to standard deviation form so that coefficients can be directly compared.

## Table 3

### Age and Exposure Model Coefficients

| Variable | Male Coef | Male St.Dev. | Female Coef. | Female St.Dev. |
|---|---|---|---|---|
| Constant | -3.99 | .118 | -3.90 | .117 |
| DNA expos. | .263 | .0125 | .188 | .0137 |
| Age65 | .0730 | .0430 | .074 | .0405 |
| Age60 | .146 | .0153 | .126 | .0162 |
| Age55 | .154 | .00859 | .143 | .00874 |
| Age50 | .155 | .00583 | .142 | .00597 |
| Age45 | .150 | .00523 | .140 | .00528 |
| Age40 | .149 | .00493 | .138 | .00496 |
| Age35 | .143 | .00480 | .139 | .00476 |
| Age30 | .143 | .00469 | .139 | .00465 |
| Age25 | .139 | .00463 | .132 | .00460 |
| Age20 | .131 | .00462 | .126 | .00458 |
| Age15 | .125 | .00463 | .120 | .00460 |
| Age10 | .117 | .00467 | .117 | .00463 |
| Age05 | .110 | .00469 | .111 | .00466 |
| Age00 | .102 | .00473 | .107 | .00471 |
| Age95 | .0962 | .00478 | .105 | .00475 |
| Age90 | .0921 | .00480 | .100 | .00478 |
| Age85 | .0837 | .00483 | .0928 | .00482 |
| Age80 | .0806 | .00487 | .0920 | .00486 |
| Age>32 | .0653 | .000817 | .0538 | .000882 |
| Sum of Squares | | | | |
| Regression | 4273.7 | | 2655.3 | |
| Error | 200.7 | | 153.0 | |
| Total | 4474.4 | | 2808.4 | |
| About Mean | 4199.7 | | 2577.1 | |

The agexx variables denote a variable which for cohort xx is

$0$ if $Age < 7$
$age - 7$ if $7 < Age < 32$
$25$ if $Age > 32$

and 0 for all other cohorts. Cohorts are denoted by the last two years of their median birth date and run from 1965 back to 1880. The Age>32 variable is 0 if Age < 32 and Age - 32 otherwise.

Table 4

Predicted Rates at Age 32

| Median Birth Year | White Males | White Females |
|---|---|---|
| 1865 | .146 | .238 |
| 1870 | .194 | .234 |
| 1875 | .222 | .311 |
| 1880 | .257 | .330 |
| 1885 | .309 | .313 |
| 1890 | .398 | .424 |
| 1895 | .419 | .441 |
| 1900 | .438 | .460 |
| 1905 | .553 | .494 |
| 1910 | .643 | .593 |
| 1915 | .907 | .693 |
| 1920* | .986 | .857 |
| 1925* | 1.224 | .969 |
| 1930* | 1.555 | 1.109 |
| 1935* | 1.482 | 1.116 |
| 1940* | 1.509 | .998 |
| 1945* | 1.690 | 1.051 |
| 1950* | 1.866 | 1.155 |
| 1955 | 1.690 | .998 |
| 1960 | 1.330 | .954 |
| 1965 | .800 | .807 |
| 1970 | .795 | .395 |

* denotes observed value for that cohort. Other values are predicted from the nearest observed value and the average ratio between age 32 and the observed value at that age.

Table 5

Population(Age 15-24) Weighted Measures of Exposure

| Year | DNA Exposure |
|---|---|
| 1890 | 3.185 |
| 1900 | 3.201 |
| 1910 | 3.194 |
| 1920 | 3.205 |
| 1930 | 3.212 |
| 1940 | 3.212 |
| 1950 | 3.225 |
| 1960 | 3.239 |
| 1970 | 3.238 |
| 1980 | 3.263 |
| 1985 | 3.276 |

* Data on population from US Historical Statistics and various issues of US Statistical Abstract. UV measures are mean of SMSA spectific measures within each state.

## Table 6

### Farming and Construction Employment

| Year | Total | Farming | Construction | Percent |
|---|---|---|---|---|
| 1880 | 17390 | 8920 | 900 | 56.5 |
| 1890 | 23320 | 9690 | 1510 | 48.0 |
| 1900 | 29070 | 11680 | 1665 | 45.9 |
| 1910 | 37480 | 11770 | 1949 | 36.6 |
| 1920 | 41610 | 10790 | 1233 | 28.9 |
| 1930 | 48830 | 10560 | 1988 | 25.7 |
| 1940 | 56290 | 9575 | 1876 | 20.3 |
| 1950 | 63377 | 9926 | 2364 | 19.4 |
| 1960 | 71489 | 7057 | 2926 | 14.0 |
| 1970 | 84889 | 4596 | 3588 | 9.6 |
| 1980 | 108544 | 3705 | 4346 | 7.4 |
| 1985 | 117167 | 2941 | 4673 | 6.5 |

* Data from 1950 are not strictly comparable to earlier data. Data from 1880 to 1940 are from US Historical Statistics, US Dept of Commerce, Washington DC 1975(Series D167, D170 and D173). Data from 1950 to 1985 are from Economic Report of the President, Council of Economic Advisors, Washington, DC, 1989(Tables B32, B43, and B98).

Table 7

Per Capita Hours of Recreational Activity

| Year | Hours |
|---|---|
| 1900 | 4 |
| 1910 | 7 |
| 1920 | 20 |
| 1930 | 43 |
| 1940 | 59 |
| 1950 | 80 |
| 1960 | 116 |
| 1970 | 211 |
| 1980 | 272 |
| 1985 | 304 |

*Data through 1960 are adapted from Clawson and Knetch, The Economics of Outdoor Recreation. From 1970 to 1985, they are extended by computing an index based on visits to National Parks, National Forests, State Parks, Personal Consumption Expenditures for Gardening, and Travel to the Carribean and South America.

Table 8

Percent Participating in Vacation
Not at Work by Reason of Vacation
(annual average, 1000's)

| Year | On Vacation | Percent |
|---|---|---|
| 1985 | 3338 | 34.2 |
| 1980 | 3320 | 36.7 |
| 1975 | 2815 | 35.4 |
| 1970 | 2341 | 33.1 |
| 1965 | | |
| 1960 | 1576 | 26.5 |
| 1955 | 1268 | 22.7 |
| 1950 | 1137 | 21.5 |
| 1946 | 662 | 13.8 |

* Percent assumes everybody counted during the year as being not at work due to vacation is distinct and multiplys not at work by 12 to get an estimate of total fraction of the work force which takes a vacation.

### Table 9

### Household Ownership of Motor Vehicles

| Year | Total | Percent One Car | Percent Two or More |
|---|---|---|---|
| 1948 | 54 | | |
| 1950 | 59 | 52 | 7 |
| 1955 | 70 | 60 | 10 |
| 1960 | 77 | 62 | 15 |
| 1965 | 79 | 55 | 24 |
| 1970 | 82 | 54 | 28 |
| 1977 | 84 | 47.5 | 36.5 |

Source: US Historical Statistics 1948-1970, Motor Vehicle Facts and Figures, 1978.

## Table 10

## Access to Motor Vehicles

| Year | Cars (1,000's) | Total (1,000's) | Per Capita | House-holds (1,000's) | Per HH |
|---|---|---|---|---|---|
| 1890 | 0 | 0 | 0 | 12690 | 0.0 |
| 1895 | 0 | 0 | 0 | 14341 | 0.0 |
| 1900 | 8 | 8 | 0.00011 | 15992 | 0.0005 |
| 1905 | 77 | 79 | 0.00094 | 17939 | 0.0044 |
| 1910 | 458 | 469 | 0.0051 | 20183 | 0.023 |
| 1915 | 2332 | 2491 | 0.025 | 22501 | 0.11 |
| 1920 | 8132 | 9239 | 0.087 | 24467 | 0.38 |
| 1925 | 17440 | 19941 | 0.17 | 27540 | 0.72 |
| 1930 | 22973 | 26532 | 0.22 | 29997 | 0.88 |
| 1935 | 22495 | 26230 | 0.21 | 31892 | 0.82 |
| 1940 | 27372 | 32035 | 0.24 | 35153 | 0.91 |
| 1945 | 25695 | 30638 | 0.22 | 37503 | 0.82 |
| 1950 | 40191 | 48567 | 0.32 | 43554 | 1.12 |
| 1955 | 51961 | 61949 | 0.37 | 47874 | 1.29 |
| 1960 | 61420 | 72887 | 0.40 | 52799 | 1.38 |
| 1965 | 74909 | 89090 | 0.46 | 57251 | 1.56 |
| 1970 | 88775 | 106808 | 0.52 | 63401 | 1.68 |
| 1975 | 106077 | 130919 | 0.61 | 71120 | 1.84 |
| 1980 | 120866 | 153358 | 0.67 | 80776 | 1.90 |
| 1985 | 129329 | 167342 | 0.70 | 86789 | 1.93 |

Table 11

Sales of Sun Protection Products
(Real 1988 $, 1,000,000's)

| Year | Total | | SPF > 8 | |
|---|---|---|---|---|
| | Sales | Ounces | Sales | Ounces |
| 1960 | 85.6 | 57.1 | | |
| 1965 | 115.8 | 77.2 | | |
| 1970 | 155.3 | 103.6 | | |
| 1975 | 193.9 | 129.3 | | |
| 1980 | 238.9 | 159.3 | 80.0 | 53.3 |
| 1984 | 249.0 | 189.5 | 103.3 | 68.9 |

Source: I am indebted to Jim Murdoch and Mark Thayer for providing the original data.

## Table 12

## Potential Mortality Impacts of Reducing Acute Exposure

### Males-1940 Birth Cohort

| Age | Baseline Mortality | Reduced Mortality | Difference | Expected Lifetime | Total Add. Yrs. |
|---|---|---|---|---|---|
| 0-4 | .28 | .07 | .21 | 70.7 | 14.85 |
| 5-9 | .26 | .07 | .19 | 65.8 | 12.50 |
| 10-14 | .51 | .20 | .31 | 60.9 | 18.88 |
| 15-19 | 1.04 | .42 | .62 | 56.1 | 35.01 |
| 20-24 | 2.05 | .82 | 1.23 | 51.4 | 63.22 |
| 25-29 | 4.06 | 1.63 | 2.44 | 46.8 | 114.00 |
| 30-34 | 8.00 | 3.20 | 4.80 | 42.2 | 202.56 |
| 35-39 | 11.27 | 4.51 | 6.76 | 37.5 | 253.58 |
| 40-44 | 15.77 | 6.31 | 9.46 | 32.9 | 311.30 |
| 45-49 | 22.39 | 8.96 | 13.43 | 28.4 | 381.53 |
| 50-54 | 30.93 | 12.37 | 18.56 | 24.2 | 449.10 |
| 55-59 | 42.02 | 16.81 | 25.21 | 20.2 | 509.28 |
| 60-64 | 55.68 | 22.27 | 33.41 | 16.6 | 554.57 |
| 65-69 | 71.09 | 28.44 | 42.65 | 13.3 | 567.30 |
| 70-74 | 85.72 | 34.29 | 51.43 | 10.5 | 540.04 |
| 75-79 | 95.00 | 38.00 | 57.00 | 8.0 | 456.00 |
| 80-84 | 93.35 | 37.34 | 56.01 | 6.0 | 336.06 |
| 85+ | 75.98 | 30.39 | 45.59 | 4.5 | 205.15 |
| Total | 615 | 247 | | | 5025 |

## Table 12 Continued

### Females-1940 Birth Cohort

| Age | Baseline Mortality | Reduced Mortality | Difference | Expected Lifetime | Total Add. Yrs. |
|---|---|---|---|---|---|
| 0-4 | .26 | .10 | .16 | 77.4 | 12.07 |
| 5-9 | .24 | .10 | .14 | 72.5 | 10.44 |
| 10-14 | .45 | .18 | .27 | 67.6 | 18.25 |
| 15-19 | .85 | .34 | .51 | 62.7 | 31.98 |
| 20-24 | 1.57 | .63 | .94 | 57.8 | 54.44 |
| 25-29 | 2.92 | 1.17 | 1.75 | 53.0 | 92.86 |
| 30-34 | 5.40 | 2.16 | 3.24 | 48.1 | 155.84 |
| 35-39 | 6.95 | 2.78 | 4.17 | 43.3 | 180.56 |
| 40-44 | 8.89 | 3.56 | 5.33 | 38.5 | 205.36 |
| 45-49 | 11.51 | 4.60 | 6.91 | 33.9 | 234.11 |
| 50-54 | 14.63 | 5.85 | 8.78 | 29.4 | 258.07 |
| 55-59 | 18.43 | 7.37 | 11.06 | 25.0 | 276.45 |
| 60-64 | 22.95 | 9.18 | 13.77 | 21.0 | 289.17 |
| 65-69 | 28.04 | 11.22 | 15.12 | 17.2 | 289.37 |
| 70-74 | 33.30 | 13.32 | 19.98 | 13.6 | 271.73 |
| 75-79 | 37.81 | 15.12 | 22.69 | 10.5 | 238.20 |
| 80-84 | 39.81 | 15.92 | 23.89 | 7.7 | 183.92 |
| 85+ | 36.05 | 14.42 | 21.63 | 5.6 | 121.13 |
| Total | 270 | 108 | | | 2924 |

## FOOTNOTES

Forthcoming at the Conference

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# Acute Health and Variable Air Pollutants

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## I. Introduction

Previous epidemiological studies [4,5,6,8] have established an empirical relationship between measures of urban air pollution (the dose) and human illness (the response). The results from these studies are interesting to policy analysts because they can, in principle, be used to predict the impacts of proposed air pollution control policies on urban populations. These aggregate dose-response predictions are credible to the extent that they seem to confirm the association between air pollutants and illness that has been found in clinical studies [2,7]. Nevertheless, the sensitivity or robustness of the predictions from the estimated dose-response functions to alternative specifications, datasets, and estimation strategies remains an important issue [2].

This paper addresses two methodological issues in estimating air pollution dose-response functions. Both concern accuracy in measuring the air pollution dose.

The first involves the intracity variation in the pollution data and the location of respondents. Previous studies have not assigned respondents different location codes over the day. Yet many people, especially those working, get a different dose during the day, when compared to the evening when they are at home. The dataset analyzed here facilitates a work and home assignment of air pollution to individuals within a city. This allows us to compare the responses of individuals to air pollution at their workplace with their responses to air pollution at home. By modeling more of the variation in the pollution data, we measure the "real-world" dose; hopefully, improving the accuracy of the estimates of the influence of air pollution on human health.

The second issue concerns the intralocation variation in air pollution. Since air pollution in an urban area can vary from hour to hour, we hypothesize that the dose is more appropriately modeled as variable over a day. Even when an individual does not change locations, he or she will experience different doses as the air pollution varies from hour to hour. Therefore, the air pollution dose depends on where and when a person is exposed. The air pollution doses used in previous studies have been based on a periodic (either one year or two weeks) average. By averaging the pollution data, the intraday variation in the data, which may influence human health, has been ignored. This can cause specification error bias, owing to left out and incorrectly measured variables in the dose-response function.

The remainder of this paper is organized into four sections. In Section II, we present a brief review of the relevant literature on estimating the relationship between air pollution and human morbidity. The empirical models, data, and basic estimation methods are described in Section III. The results are presented in Section IV, while the last section contains concluding remarks.

## II. Air Pollution and Morbidity: Previous Studies

The design of this paper is most closely related to the studies by Ostro [4,5], Hausman et al. [3], and Portney and Mullahy [6]. These authors use the Health Interview Survey (HIS), an annual health survey of people in various locations throughout the U.S., to study the empirical relationship between air pollution and human morbidity. Morbidity is measured by a variable that reflects the changes in the normal activities, owing to health impairments, of the survey respondents during the 2-week recall period of the HIS. Several pollutants are analyzed, including measures of the atmospheric concentrations of total suspended particulates (TSP), ozone, fine particulates, and sulfates. In addition to including several socio-economic and weather measures in their models, these authors examine numerous subsamples based on sex, working status, and smoking status, attempting to hold constant as many confounding influences as possible.

In Ostro [4], the variation in the air pollution data comes from the pooling of respondents from different cities. Doses were measured by the annual average of total suspended particulates (TSP) and sulfates (SO4) and ignore the intrayear and intraday variation in the pollution data. The morbidity measures reflect the number of "work loss days" (WLD) and the number of "restricted activity days" (RAD) that survey respondents reported during the two-week recall period. The TSP term is significant and has the expected relationship to RAD and WLD. The SO4 term is not significant, which may not be surprising since SO4 is more localized than TSP.

In his follow-up piece, Ostro [5] uses a Poisson distribution to model the relationship between the number of RADs and the contemporaneous (with the survey) two-week average of fine particulates. Fine particulates are estimated from airport visibility and TSP data. The two-week average of fine particulates is significant over several different samples and years.

Hausman et al [3] concentrate on WLD and specifically control for intrayear variation in pollution. Additionally, they estimate models with alternative lags of the two-week (in contrast to the annual) average of TSP, although no formal tests to choose among the specifications are presented. Like the Ostro studies, intracity variation in the pollution exposure (within a period) is ignored and the SO4 measure is not significant. Hausman et al provide some empirical support for the Poisson specification; i.e., the pollution coefficients were robust when the Poisson assumption that the variance equal the mean was relaxed and when a fixed effects model was estimated.

Portney and Mullahy [6] estimate a Poisson model with the number of respiratory related RAD as the dependent variable and various measures of ozone and sulfates for the exposure variables. Portney and Mullahy's ozone exposure measures are probably better suited to test acute health effects because they average over the daily maximum of ozone during the two-week recall period. Moreover, by matching respondents to the pollution monitoring stations closest to their census tract (and within a 10 and a 20 mile radius of their census tract). Portney and Mullahy analyze some of the within city variation in the pollution data. They find that the estimates for the ozone coefficients do not vary greatly among these different assignment strategies, meaning that the intracity variation is not empirically important in their data. As in the aforementioned studies, sulfates do not perform in an *a priori* expected manner.[1]

## III. Methodology and Data

The methodology and data used in this study were constructed in order to examine the robustness of acute health predictions. In particular, we propose to compare the predicted health responses from a "traditional" specification to specifications where the pollution measures and assignments more accurately reflect real world exposures. This comparison exercise will provide information for policy makers as well as future research efforts.

Define the following notation:

- $H_{it}$ = the health response of individual i in time period t.
- $X_i$ = a vector of individual specific covariates.
- $W_t$ = the weather in time t.
- $POL_{it}(L)$ = the pollution exposure experienced by i in time period t. The exposure is a function of i's location (L) over the time period.

Then,

$$H_{it} = f(X_i, W_t, POL_{it}(L)) \qquad (1)$$

is a hypothesized dose-response function.

To estimate a model like (1), requires data on $H_{it}$, $X_i$, $W_t$, and $POL_{it}(L)$ and a functional form for the model. The necessary data were obtained from a health survey, the Weekly Weather and Crop Bulletin, and the SAROAD system data tapes. The functional form for the model was specified to be consistent with previous studies.

### The health survey data

During 1978-1980, Geomet Technologies, Inc. administered a health survey to the members of 2,594 households in the greater St. Louis area. Households were enrolled in the survey in groups of about eighty per week beginning June 4, 1978 and ending May 27, 1979. The respondents maintained daily logs of their activities, locations, restrictions in activities, and the reasons for any restrictions in activities. The logs were kept over four two-week periods; thus, the dataset includes the restrictions on activities and the locations for each respondent for 56 days.

The structure of the survey also facilitated the collection of extensive data on socioeconomic conditions, lifestyle choices, work environment, medical care, and health.[2] A complete description of the data and the datafiles are available from the authors upon request.

The appropriate measure of the health response depends on the focus of the study. Here, we are particularly interested in acute respiratory health responses. As an empirical measure of

$H_{it}$, we used the number of RADs reported by the respondent in the time period, owing to a respiratory disorder or symptom ($NRRAD_t$). Given this type of limited dependent variable, a reparameterization of the Poisson distribution is a particularly attractive statistical model for equation (1).

As shown elsewhere [6], the expected value of $NRRAD_t$ under the Poisson model is given by

$$E(NRRAD_t) = \exp(X_i\beta + W_{it}\gamma + POL_{it}(L)\delta) \quad (2)$$

where the $\beta$, $\gamma$, and $\delta$ represent parameter vectors that are estimated via maximum likelihood methods. Using equation (2), a prediction for a small change in a $POL_{it}(L)$ variable (or any other) is a straightforward computation.

The variables in $X_i$ should include measures on i's age, income, living arrangements, working conditions, personal health habits, and personal health status. Since an incorrect specification of the $X_i$ could bias the estimates of the relationship between $H_{it}$ and $POL_{it}(L)$, we included several covariates. Moreover, the data were limited to people between the ages of 16 and 65 who are non-smokers and working outside of the home.[3]

A brief description and summary statistics of the $X_i$ covariates, the weather covariates, and NRRAD are presented in Table 1.

Pollution Data

The pollution data were obtained from the U.S. Environmental Protection Agency's SAROAD system. The data tapes contain hourly observations, collected at 14 monitoring sites, on numerous pollutants in the St. Louis Air Quality Control Region.[4] The pollution data were matched to the survey respondents by time, as described below, and location vis-a-vis the monitoring stations. The respondents averaged about three miles from a monitoring site. The pollutants analyzed here, ozone and sulfur dioxide (SO2), were chosen for two reasons. First, the data for these two pollutants were collected at all of the monitoring stations over the time period of the health survey. For the other air pollution measures, for example, total suspended particulates and NO2, the data are not available for several weeks during the survey period or they were not collected at each site. Second, SO2 tends to be more localized than ozone. This contrasting nature of the two pollutants provides a natural "laboratory" for measuring the appropriateness of our measures and assignments of pollution.

The pollution measures differ from the $X_i$ and the $W_t$ because an individual's exposure to pollution is not constant over t. Pollution exposure can vary as individuals change locations over the day. Even when an individual is stationary, their exposure changes as the pollution varies over the course of the day.[5]

In defining the measures of air pollution dose, our objective was to preserve as much variation in the pollution data as possible. The format of the health survey means that, for each enrollment week, there are four associated two-week periods. Since we used the survey data from weeks 1 through 41, there are

164 two-week periods. However, within each two-week period, we grouped the data into "day-time" observations (the hours 11:00 am through 5:00 pm) and "night-time" observations (the hours 5:00 am through 10:00 am and 6:00 pm through 12:00 pm).[6] The pollution data were, therefore, initially grouped into 328 subperiods. Each day-time subperiod contains 98 observations, while the night-time subperiods consist of 182 observations.

For each subperiod, we computed the following sets of parameters:

(i) The mean and standard deviation. If the data are normally distributed, then these parameters fully describe the distribution of the pollution.

(ii) The mean and standard deviation of the natural logarithm of the data. If the data are lognormally distributed, which may be more plausible than normality, then these parameters characterize the distribution of the dose.

Also, to facilitate a comparison with previous models, the two-week mean and the average over the daily maximums were computed.

Two methods were developed for investigating the sensitivity of the dose-response function to the individual's pollution exposure. Both address the variability in air pollution doses.

## The impact of locations changes on pollution measures

For an individual who lives in one location and works in another, we are uncertain about the correct assignment of the pollution dose. With the data analyzed here, each respondent has two location codes; a home code and work code. The home location pollution, the work location pollution, or some combination of the two could seemingly be used to assign pollution exposures to the individuals. Moreover, the pollution data reflect different times of the day; thus, the home code may be more appropriate for night-time exposures and the work code more appropriate for day-time exposures. Since we are uncertain about the correct assignment, one possibility is to let the data determine it.

Let $\theta_1$ be the fraction of the total exposure time to air pollution experienced during the day at work. Similarly, let $\theta_2$ be the fraction experienced at home during, our definition of, the night-time. Finally, let $\theta_1$ be the fraction of exposure time experienced at home during the day. We assume that $\theta_1 + \theta_2 + \theta_3 = 1$, implying that all of the exposure is experienced in the manner hypothesized.

The $\theta$´s, if assumed to be unknown, can be estimated given some criterion. the correct mean exposure experience by a respondent is a weighted average of the means at each location for each time period, where the weights are the $\theta$´s. Let

WDPOLMU = the mean of pollution calculated from the day-time data at the work location code,
DPOLMU = the mean pollution calculated from the day-time data at the home location code, and
NPOLMU = the mean pollution calculated from the night-time data at the home location code.

Then,

$$\text{POLAVE} = \theta_1\text{WDPOLMU} + \theta_2\text{DPOLMU} + \theta_3\text{NPOLMU} \qquad (2)$$

is the weighted average pollution experienced by the respondent. We use a grid search over the $\theta$´s to find the set that maximizes the likelihood function.

As reference points, we alternatingly let each of the $\theta$´s have a value of 1. Additionally, we assumed 112 hours of exposure per week; 40 at work, 56 at home during the night-time hours, and 16 at home during the day-time hours. These assumptions give $\theta_1 = 40/112$, $\theta_2 = 56/112$, and $\theta_3 = 16/112$.

The impact of intraday variation on pollution measures

Some intraday variation in the pollution data is captured by using the day-time and night-time means. However, there does not seem to be any theoretical reason for using the mean of the pollution distribution to measure the dose. In fact, a simple example illustrates that using the mean imposes a linear restriction on the dose-response function. Assume that the pollution exposure for some individual is variously POL1, POL2, and POL3 over the time period. Let the probability of each level occurring be f1, f2, and f3, respectively. Then, the mean equals f1*POL1 + f2*POL2 + f3*POL3. Next, let the dose-response function be linear in parameter space. Write it as

$$H = \delta_0 + \delta_1(f1*POL1) + \delta_2(f2*POL2) + \delta_3(f3*POL3)$$

for some individual in some time period. If $\delta_1 = \delta_2 = \delta_3$, then using the mean is equivalent to entering the probability distribution. On the other hand, different $\delta$´s would indicate that a mean model incorrectly restricts the health response to changes in average pollution; i.e., the response depends on how the mean changed. The problem with this method is that we have no guidance on the number of δ's to specify. Still, this type of analysis may provide insights into the health response of distributions of air pollution.

## IV. Empirical Results

### The impacts intraday variability in pollution exposures

In Table 2 we display the parameter estimates from several models. Only data from the first follow-up period was used to estimate the parameters. While the qualitative conclusions are similar for the second follow-up period, we could not statistically pool the data from the first two periods. The estimates based on the third and fourth period observations were quite different than those obtained from the first two. In particular, most of the parameter estimates were sensitive to the alternative specifications. This problem is apparently symptomatic of some type of survey bias, perhaps because respondents lost interest after the first two periods.

The specifications presented in Table 2 differ in the type of pollution measures entered. Several other independent variables could be selectively entered into the specifications. Those presented here are representative of the literature. The pollution coefficients are not particularly sensitive to any of the measures, except the seasonal dummies and the weather variables. Selectively dropping these variables can change the sign of the pollution measures in some of the specifications. The weather variables and the seasonal dummies are statistically significant in each specification, however.

The first specification represents a "traditional" air pollution dose-response function. The air pollution is assigned to the respondents' home location code. The variable labels represent average ozone (OZMU) and average SO2 (SOMU), where the average is computed using data from the entire day. Fourteen of the eighteen coefficient estimates exhibit p-values of less than .05.[7] As in other studies using a sulfur-oxide term, SOMU has a negative sign but is insignificant.

The remaining coefficient estimates are remarkably stable across different specifications. They show, all else equal, that:

- older respondents have fewer expected restricted activity days due to respiratory symptoms and disorders [E(NRRAD)],
- years of education do not affect E(NRRAD),
- the E(NRRAD) is lower for males,
- respondents in higher income classes have a lower E(NRRAD),
- respondents with good perceived health have a lower E(NRRAD),
- previous smokers have a greater (or insignificant) E(NRRAD),
- cooler temperatures and more rain increase E(NRRAD),
- when respondents are exposed to irritants at work, E(NRRAD) diminishes,
- respondents who exercise regularly, reduce E(NRRAD), and
- the greatest E(NRRAD) occurs during weeks 17-24, which is from the end of September to the middle of November.

One of the most interesting estimates is the coefficient on EXER. If individuals can reduce their expected number of respiratory related activity days by "expenditures" on regular exercise, this may give analysts an avenue for assessing the benefits of a cleaner environment.

Specifications (2), (3), (4), (5), and (6) illustrate the impacts of using different assignment methodologies (or weights) for the pollution terms. As indicated above, the actual dose is hypothesized to be some combination of the air pollution at home during the day (DOZLMU and DSOLMU), the air pollution at home during the night (NOZLMU and NSOLMU), and the air pollution at work during the day (WDOZLMU and WDSOLMU).

We used the mean of the natural logarithm of ozone and SO2 (all variable end with "LMU") because the log of the data appeared to be more normally distributed than the levels; thus, using the mean of the logs is a better measure of the central tendency in the data.[8]

The impacts of changing the $\theta$'s are dramatic on the estimated coefficients for ozone and SO2. In specification (2), $\theta_1 = \theta_3 = 0$ and $\theta_2 = 1$. The respondents are assigned the mean pollution at their homes computed over the day-time hours (11:00 am - 5:00 pm). The coefficient on DSOLMU remains insignificant, but becomes positive, and the likelihood function rises (the negative falls) slightly. Since the maximum reading usually occurred during this time interval, this specification is similar to [6], who used the average of daily maximums.

The third specification shows that, when the respondents are assigned a dose based on their home location pollution average over the night-time hours (5:00 am - 10:00 am and 6:00 pm - 12:00 pm), the ozone influence remains stable and the SO2 coefficient remains insignificant. The SO2 coefficient estimate jumps noticeably in magnitude, however, and the likelihood function continues to rise.

$\theta_1 = 1$ and $\theta_2 = \theta_3 = 0$ in specification (4). The SO2 term is significant and of similar size to the one exhibited in (3). The coefficient estimate on the ozone term is also significant and about the same size as the estimate in (2) and (3).

Specifications (5) and (6) show the impact of non-zero $\theta$´s. The $\theta$´s are constructed a priori in specification (5), while in six they are estimated using a grid search. The log of the likelihood functions are the same up to the second decimal point. When comparing the coefficient estimates in (5) and (6) to the estimates obtained with the other models, we see that the impact of the pollution terms increases as the measures approach "real world" exposures.

The empirical significance of the alternative models is displayed in Table 3. Predictions of the expected value of NRRAD from each specification for one thousand identical individuals are shown. The predictions differ by the type of change in ozone and SO2.

A change in just ozone (Predict2) can change the prediction on E(NRRAD) by from 1.928 per thousand to 10.671 per thousand; a difference of over 400%. Similarly, a change in just SO2 (Predict3) can change the E(NRRAD) per thousand from .356 (or -2.393, using specification 1) to 9.246; over 2000%. Clearly, the choice of the pollution measure can have a dramatic impact for policy analysts.

## An analysis of non-mean models

As noted above, it is possible to test the mean specification. Based on the mean and standard deviation estimates of the logged data and assuming both pollutants were lognormally distributed, we computed the probability that the pollutants would fall into various categories. For ozone, we chose four categories; 0-5, 5.01-20, 20.01-60, and greater than 60. For SO2, we used 0-5, 5.01-10, 10.01-25, and greater than 25. The probabilities were computed for each of the distributions used above (i.e., day-time work, day-time home, and night-time home) and then averaged using the maximum likelihood estimates for the $\theta$'s. The specifications with the probabilities entered as dependent variables did not significantly improve the model.[9] This was true for both ozone and SO2, indicating that the exposure time weighted mean model can not be rejected. Evidently, the distributional aspects of the air pollutants are adequately captured by specification (6) for the data analyzed here.

## V. Conclusions

The primary conclusion of this paper is that predictions from alternative estimated dose-response functions differ substantially, depending on how pollution exposures are measured and assigned to individuals. The exposure varies because individuals' locations and air pollutants are not constant throughout a day. Dose-response specifications that use a weighted average of pollution experienced during the day at home, during the day at work, and during the night at home statistically outperform more traditional models. Moreover, the weighted average models indicate that the pollutants adversely affect human morbidity more than traditional models.

Our results indicate that sulfur-dioxide adversely affects human health. This finding is different from previous studies. The apparent reason for the difference is our treatment of the variable nature of the pollution. This particularly appealing, in the case of SO2, because SO2 is more localized than ozone. Hence, too much aggregation in the pollution data would mask the strength of the influence. By disaggregating the data, we have, hopefully, uncovered the true relationship.

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## Endnotes

1. Portney and Mullahy find nonlinearities in the dose-response function. The marginal responses to increases in ozone are greater when equations are estimated with the data reflecting higher (greater than .05 pphm) ozone concentrations. They also find that the elasticity of the expected value of the respiratory RAD is not constant with respect to changing ozone as implied by the simple poisson distribution.

2. Survey respondents provided information on the number of visits to a doctor, the travel time to the doctor, visits to emergency rooms, and other "cost" measures. Gerking and Stanley [ ] use these data to estimate a willingness to pay for reduced air pollution expression that is based on an averting behavior model of consumer choice.

3. A statistical test indicated that the smokers could not be pooled with the non-smokers using a dummy variable to reflect smoking status.

4. An independent benchmark, the Regional Air Monitoring System (RAMS) data, was used to assess the quality of the SAROAD data. The RAMS data, which were collected in the late 1970s, but not during the time of the health survey, were subjected to extensive quality control and more accurately measure airborne pollutants. During the time that both systems were operational, the ozone readings between RAMS and nonRAMS data exhibit zero order Pearson correlations in the range of .3 to .6 for the hourly data. These correlations improve substantially as the data are aggregated to the daily and weekly level. Hence, there is every reason to expect that our use of the SAROAD data over a two-week period accurately measures the air pollution dose.

5. We did not model the weather as changing over the course of the day. It probably should be. However, the methodology proposed here facilitates a comparison to previous studies.

6. We computed Chi-square tests of distributional independence for all possible aggregations of the daily data by monitoring station. In the vast majority of cases, we cannot reject the hypothesis that the 11 am - 5 pm data come from the same distribution. Similarly, we cannot reject the hypothesis that the 5 am - 10 am and 6 pm - 12 pm data come from the same distribution. The hypothesis that all the daily observations are generated by the same distribution was rejected in most cases, however.

7. The p-values are based on the variance-covariance matrix computed directly from the maximum likelihood estimates. They do not reflect a correction like in [6] or [3].

8. To identify the appropriate distribution of the data, we estimated the "transformed" mean and standard deviation and the transformation parameter. These parameters are based on the powernormal distribution, which utilizes the Box-Cox power transformation. The Box-Cox transformation facilitates a test between normal and lognormal distributions. In the majority of cases for SO2, the lognormal distributional assumption could not be rejected. With respect to ozone, we found that, usually, both the normal and lognormal distributions could be rejected. However, the transformation parameter was closer to 0 (indicating lognormal), than to 1.

9. We tested the probability model three ways. Firstly, by entering the probabilities for just ozone. Then, by entering the probabilities for just SO2, and, finally, by entering both. None of the Chi-squares, comparing twice the difference in the log likelihood values, indicated rejecting the linear constraint imposed by the mean specifications.

## Table 1.
### Variable Descriptions and Summary Statistics
### Non-smokers, First Follow-up Period.
### (Observations = 597)

| Variable | Description | Mean | StDev. | Minimum | Maximum |
|---|---|---|---|---|---|
| AGE | Age in years | 38.88 | 13.37 | 18 | 65 |
| EDUC | Years of school | 13.02 | 2.93 | 0 | 24 |
| SEX | 1 if male | .49 | .50 | 0 | 1 |
| WHITE | 1 if white | .78 | .43 | 0 | 1 |
| INCOME | Income category | 6.06 | 1.48 | 1 | 8 |
| PHEALTH | 1 if perceived health good | .93 | .30 | 0 | 1 |
| PSMOKE | 1 if previous smoker | .18 | .39 | 0 | 1 |
| TEMP | Average temperature | 60.40 | 20.84 | 13 | 83 |
| RAIN | Average rainfall | .63 | .57 | 0 | 2.15 |
| IRR | 1 if irritants at work | .34 | .47 | 0 | 1 |
| EXER | 1 if exercise regularly | .11 | .31 | 0 | 1 |
| S1 | 1 if weeks 1 - 8 | .26 | .44 | 0 | 1 |
| S2 | 1 if weeks 9 - 16 | .30 | .46 | 0 | 1 |
| S3 | 1 if weeks 17 - 24 | .13 | .34 | 0 | 1 |
| S4 | 1 if weeks 25 - 32 | .17 | .38 | 0 | 1 |

| | |
|---|---|
| NRRAD | Number of respiratory related restricted activity days during follow-up period two. |

Frequency for NRRAD

| Value of NRRAD | Frequency |
|---|---|
| 0 | 558 |
| 1 | 39 |
| 2 | 9 |
| 3 | 7 |
| 4 | 6 |
| 5 | 2 |
| 6 | 3 |
| 7 | 4 |
| 8 | 1 |
| 9 | 0 |
| 10 | 0 |
| 11 | 1 |
| 12 | 0 |
| 13 | 0 |
| 14 | 0 |

Mean = .241

## Table 2.
## Alternative Coefficient Estimates of the Dose-Response Function
Dependent Variable = NRRAD

| Variable | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| AGE | -.033* | -.032* | -.036* | -.030* | -.034* | -.033* |
| EDUC | -.018 | -.024 | -.015 | -.024 | -.021 | -.022 |
| SEX | -.083* | -.121 | -.048 | -.118 | -.060 | -.074 |
| WHITE | .617* | .676* | .798* | .734* | .704* | .692* |
| INCOME | -.203* | -.199* | -.188* | -.164* | -.192* | -.191* |
| PHEALTH | 1.146 | 1.266* | 1.201* | 1.326* | 1.268* | 1.284* |
| PSMOKE | .219* | .238 | .181 | .129 | .185 | .188 |
| TEMP | -.054* | -.042* | -.038* | -.037* | -.040* | -.041* |
| RAIN | .853* | .957* | .716* | .956* | .982* | 1.031* |
| IRR | -.302* | -.319* | -.214 | -.273 | -.229 | -.245 |
| EXER | -.888* | -.863* | -.800* | -.819* | -.854* | -.861* |
| S1 | -2.441* | -2.681* | -2.321* | -2.536* | -2.867* | -2.941* |
| S2 | 1.318* | .773 | 1.421* | .796* | .853* | .719* |
| S3 | 1.565* | 1.268* | 1.575* | 1.107* | 1.516* | 1.446* |
| S4 | .841* | 1.036* | 1.057* | 1.086* | 1.410* | 1.424* |
| OZMU | .037* | | | | | |
| SOMU | -.028 | | | | | |
| DOZLMU | | .951* | | | | |
| DSOLMU | | .065 | | | | |
| NOZLMU | | | .989* | | | |
| NSOLMU | | | .407 | | | |
| WDOZLMU | | | | .905* | | |
| WDSOLMU | | | | .367* | | |
| OZLAVE1 | | | | | 1.692* | |
| SOLAVE1 | | | | | .587* | |
| OZLAVE2 | | | | | | 1.708* |
| SOLAVE2 | | | | | | .571* |
| CONST | 1.043 | -2.442* | -2.858* | -3.568* | -5.560* | -5.705* |
| $\theta_1$ | na | 0 | 0 | 1 | 40/112 | .4 |
| $\theta_2$ | na | 1 | 0 | 0 | 56/112 | .4 |
| $\theta_3$ | na | 0 | 1 | 0 | 16/112 | .2 |
| -L like | 270 | 269.5 | 268.8 | 269.4 | 264.5 | 264.5 |

*Indicates that the p-value is less than .05.

**Table 3.**
**Predicted Reductions in the Expected Value of NRRAD Per 1000 People by Specification**

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| Predict1 | .637 | 2.197 | 14.294 | 5.418 | 16.351 | 12.940 |
| Predict2 | 2.290 | 1.928 | 8.188 | 3.089 | 10.671 | 8.546 |
| Predict3 | -2.393 | .356 | 8.119 | 3.022 | 9.246 | 7.186 |

Notes: The predictions are based on the following initial values: AGE=40, EDUC=12, SEX=1, WHITE=1, INCOME=6, PHEALTH=1, PSMOKE=0, TEMP=70, RAIN=.5, IRR=0, EXER=0, S1=1, S2=0, S3=0, and S4=0. The initial value for ozone is 40, while the initial value for SO2 is 20. The predictions are per 1000 people, where:

Predict1 is based on reducing ozone to 30 and SO2 to 10,
Predict2 is based on reducing ozone to 30 and maintaining SO2,
Predict3 is based on reducing SO2 to 10 and maintaining ozone.

# Pricing Environmental Health Risks:
# Survey Assessments of
# Risk-Risk and Risk-Dollar Trade-offs*

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# Abstract

This study develops a methodology for measuring the values that individuals place on morbidity risk reductions and applies it to the measurement of the benefits from reducing the risks of contracting chronic bronchitis. The survey methodology involves the use of an iterative computer program that presents respondents with a series of pairwise comparisons which are individually designed to measure respondents' marginal rates of substitution for chronic bronchitis risk reduction. The approach is innovative in that it measures the rates of trade-off for chronic bronchitis risk reduction in terms of the risk of an automobile accident fatality, as well as in dollars. Since it generates estimates for each individual, it can reveal distributions of benefit measures rather than simply a population mean estimate. The resulting rates of trade-off for chronic bronchitis and auto fatality risks suggests that the risk of a chronic bronchitis case is worth 32% of the comparable risk of death, as measured by the median trade-off rate. When risk reduction for chronic bronchitis is compared to a cost of living increase, the median rate of trade-off is $457,000, whereas the comparison between automobile fatality risk reductions and cost of living increases yielded a median rate of trade-off of $2.29 million. The results across different risk-risk and risk-dollar trade-offs were internally consistent.

## 1. Introduction

Over the past decade economists have devoted substantial attention to the implicit valuation of health outcomes. These analyses of risk-dollar trade-offs have relied in large part on market-based data.[1] For example, wage-risk trade-offs have been used to analyze the implicit value of fatalities and the average nonfatal job accident risk. Similarly, economists have analyzed the trade-offs implied by seat-belt usage decisions to infer a value of life.[2]

Although studies using market data provide useful benchmarks for health risk valuation, they do not resolve the issue of how government agencies should attach benefit values to health outcomes for which we do not have good market data. This omission is particularly important for government agencies, such as the U.S. Environmental Protection Agency (EPA), which generally focus on policy contexts in which market forces are believed to not be fully effective. For these situations, no useful market trade-off data may be available. Nevertheless, economic analysts would like to select the efficient project mix, and some benefit measure is required to perform such an analysis. In recent years, a large number of studies have addressed these benefit issues using non-market techniques, thus greatly expanding the range of benefit components that can be valued.[3]

This study makes several contributions to the literature on non-market techniques for benefit valuation. First, we develop a methodology for measuring the benefits of reducing the risks from various types of morbidity effects. The methodology uses an iterative computer program to ascertain the points of indifference for consumers who are asked to trade off the reduced morbidity risk with increases in other attributes of a location decisions, such as an area's cost of living and the risk of an automobile fatality.[4]

Second, we apply the methodology to an important health benefit valuation problem, that of estimating the value of reductions in the risk from chronic bronchitis, one central type of chronic obstructive pulmonary disease alleged to be a major adverse effect of ozone pollution exposure. Most previous studies of health valuation focus on acute health effects, such as accidental death, rather than chronic diseases whose effects are more difficult to communicate to potential victims.[5]

Third, our approach yields the entire distribution of consumer values for chronic bronchitis risk reduction, rather than just the mean valuations which can be derived from market-based approaches to the problem. This information is important for policy makers in situations where consumers place widely divergent values on reducing risk.

Fourth, because chronic disease effects are difficult to communicate to potential sufferers, it is important to use a methodology that adapts to whether subjects understand the valuation task being asked of them. By administering the questionnaires interactively on a computer, our approach allows us to build in several tests of task comprehension that, if failed, provide additional information before proceeding with the questionnaire.

Finally, our methodology produces values for morbidity risk reduction in terms of trade-offs with several other metrics besides money. In our chronic bronchitis application, we measure trade-offs with the risk of automobile fatalities, as well as with a dollar measure derived from changes in the cost of living. Many policy-makers are hesitant to base decisions on benefits denominated in dollars, and they may be more willing to implicitly consider benefit values when measured in units of a common risk such as death. Converting all health outcomes into death risk equivalents facilitates cost-effectiveness analysis by calculating the cost per statistical life equivalent saved, and it addresses concerns with respect to dollar pricing. Even if the morbidity valuations are elicited in terms of trade-offs between risks, they can still be converted into dollar values by using hedonic measures of the value of the comparison risk if that comparison risk is death (with the appropriate application of sensitivity analysis to the assumed values of life used to make the translation).

There are reasons to suspect that consumers may have fewer difficulties with the task of specifying rates of trade-off of one risk with another, as opposed to trading off a risk with a certain dollar amount. The risk-dollar trade-off task sometimes produces alarmist responses from subjects who cannot envision that they would voluntarily subject themselves to a higher risk of a serious morbidity effect for a finite amount of additional income.[6] Dollar valuation tasks also are difficult to design in ways that subjects will find analogous to real choice situations, and they may offer biased responses to questions that do not force them to pay for the risk reduction being valued. There is a final reason to prefer the risk-risk trade-off approach. To the extent that consumers are equally adverse to the risks from different types of risks, asking them to trade off one risk against another produces rates of trade-off which measure the relative value to them of the two risks without regard to the risk aversion which enters in trading off uncertain health risk with certain dollars. In this sense the risk-risk trade-offs provide values which are not as heavily influenced by the consumers' attitudes towards facing risks per se.

The outline of this paper is as follows. Section 2 provides an overview of the study design and the sample. Section 3 describes the risk-risk trade-offs whereby respondents put their chronic morbidity valuations into auto death equivalents. In Section 4 we describe the direct estimates of risk-dollar trade-offs for chronic bronchitis obtained by asking respondents to trade off chronic bronchitis risks with either the area's cost of living or property damage from storms. As a check of the validity of the approach, we provide evidence on auto fatality risk-dollar trade-offs in Section 5. These implicit value of life numbers are tested against those in the literature to assess the valibity of the survey approach. In Section 5 we also convert all of our results for the value of chronic bronchitis to dollar equivalents. Section 6 concludes the paper.

## 2. Study Design and Sample Description

### General Approach

We used a sample of 593 shoppers from a blue-collar mall in Greensboro, North Carolina to measure willingness-to-pay values for reducing the probability of contracting chronic bronchitis. The subjects made four series of pairwise comparisons of different locations where they could live with the locations differing in two attributes. In most of these comparisons, one of the locational attributes varied was the probability of contracting chronic bronchitis.

The first series of questions yielded a rate of trade-off between decreases in the risk of chronic bronchitis (CB) and increases in the risk of an automobile fatality, thus providing what we call a "risk-risk" trade-off. The second series of questions determined a "risk-dollar" trade-off, where the reduction in the risk of CB was achieved at the expense of a location with a higher cost of living.

If subjects were found to more easily trade off a reduced CB risk with a higher auto fatality risk than with a cost of living increase, we wanted to sort out whether this result was due to the fact that the cost-of-living differences were measured in dollars or that they were given with certainty (that is, with no risk involved over dollar gambles). Thus, our third series of questions asked subjects to trade off reductions in the CB risk with increases in a lottery on dollar losses expressed as a risk of storm damage, where if a storm were to occur, it would cause $2,000 of damage to the subject's home and belongings. Finally, in order to compare the CB risk--auto fatality risk trade-offs with the risk-dollar trade-offs, it was useful to obtain a dollar measure of the value of reducing the risk of automobile fatalities. This fourth series of questions provided a rate of trade-off of risk reduction in automobile fatalities to increases in a location's cost of living.

The results from these four series of questions allows us to address the following questions:

* What is the distribution of CB risk--death risk trade-offs?
* What is the distribution of CB risk--(certain) dollar trade-offs?
* What is the distribution of CB risk--(uncertain) dollar trade-offs?
* Which of these three trade-offs is easier to elicit accurately from consumers?
* What is the distribution of death risk--(certain) dollar trade-offs?
* How does the distribution of CB risk--(certain) dollar trade-offs compare with the distribution of CB risk--dollar trade-offs derived from combining the CB risk--death risk trade-offs with the death risk--(certain) dollar trade-offs?
* How does the distribution of CB risk--(certain) dollar trade-offs compare with the distribution of CB risk--dollar trade-offs derived from combining the CB risk--death risk trade-offs with the values of life derived from wage hedonic studies?

It should be noted that the first question is the most important one to answer because it addresses the use of an alternative metric to dollars for measuring morbidity risk willingness-to-pay values, that of another health risk, namely death. For cost-effectiveness purposes, it is not necessary to go beyond the death risk metric, as alternative policy initiatives can be compared on the basis of this metric rather than dollars. However, if the CB risk values measured in death risk units translate closely to the direct dollar valuations of reducing CB risks that we obtain, policy makers can be more confident in the reasonableness of the risk-risk valuations.

In order to understand our empirical results that allow responses to the questions above, it is first necessary to carefully describe the design of the survey and sample.

## Methodology

The task of eliciting individuals' valuation of chronic bronchitis is not straightforward. The first problem is that few individuals fully understand the health effects of chronic bronchitis. Second, once given this information, they may not have sufficient experience in dealing directly with such trade-offs to give meaningful valuation responses. To accommodate these difficulties, we developed an interactive computer program that would inform consumers as well as elicit trade-off information.

Three different questionnaires were used, but for concreteness let us focus on what we will designate Questionnaire A. After acquainting the respondent with the computer, the program elicits information regarding the respondent's personal characteristics (e.g., age). A substantial portion of the questionnaire (about 40 questions) is then devoted to acquainting the respondent with the health implications of chronic bronchitis and the nature of the trade-offs that would be encountered. These questions elicit the respondent's familiarity with chronic bronchitis, information on smoking history, and provide a detailed summary of the health implications of chronic bronchitis.

The thirteen principal health implications of chronic bronchitis are summarized in Table 1. The chronic bronchitis disease classification includes a variety of illnesses of differing severity. Our intent was not to value each possible combination of systems, but rather to establish a methodology that could be used to value this and other adverse health effects. Consequently, our valuation procedure pertains to the set of symptoms summarized in Table 1, but the broader purpose of our analysis is to develop a methodological approach that is more generally applicable to other patterns of chronic bronchitis, as well as to different diseases such as cancer.

Since chronic bronchitis takes many forms, this study focused on the most severe chronic morbidity effects.[7] Thus, the survey's focus is on the adverse health outcomes at the extreme and of the cluster of diseases within the chronic bronchitis grouping. Because a quick overview of these effects may not be fully comprehended by respondents, in each case subsequent questions ascertain the respondents' assessed disutility ranking of each outcome in a linear 49-point scale. The purpose of these questions is not to establish attribute-based utilities, but to

## Table 1

## Health Implications of Chronic Bronchitis

1. Living with an uncomfortable shortness of breath for the rest of your life.
2. Being easily winded from climbing stairs.
3. Coughing and wheezing regularly.
4. Suffering more frequent deep chest infections and pneumonia.
5. Having to limit your recreational activities to activities such as golf, cards, and reading.
6. Experiencing periods of depression.
7. Being unable to do the active, physical parts of your job.
8. Being limited to a restricted diet.
9. Having to visit your doctor regularly and to take several medications.
10. Having to have your back mildly pounded to help remove fluids built up in your lungs.
11. Having to be periodically hospitalized.
12. Having to quit smoking.
13. Having to wear a small, portable oxygen tank.

encourage respondents to think carefully about the health implications of chronic bronchitis and their own view of the effect of this disease on their well-being.

At this point in the questionnaire, the respondents confront the first of two set of trade-off questions. Individuals are presented with a choice of moving to one of two alternative locations which differ in terms of their chronic bronchitis risk and automobile accident risk. To ensure that respondents would be willing to consider making such a move at all, they were told that these two locales posed a lower risk of both outcomes than their current place of residence.

Since risk levels differ across individuals, the program elicits information regarding individual activities that are likely to influence their person-specific risks, such as smoking habits (for chronic bronchitis) and mileage driven per year (for auto accident deaths). The program then informs the respondents that the probabilities presented in subsequent questions are calculated based on their responses to the earlier risk-related activity questions, even though the same risks are actually presented to all subjects.[8] This procedure increases the extent to which the stated risk levels are taken at face value, while facilitating the comparison of risk trade-offs across subjects because they all responded to the same risks.

To ensure that respondents understand the task before proceeding to questions in which one location is lower in one risk but higher in the other risk, they are first presented with a dominant choice situation. Let the notation (x,y) denote a locale where the chronic bronchitis probability is x/100,000 and the automobile death risk is y/100,000. The actual survey did not present the choices in such abstract terms, but this notation makes the exposition of the survey structure simpler.[9]

To ascertain whether respondents understand the task, they are first asked whether they prefer Area A with risks per 100,000 population of (75, 15) or Area B with risks (55, 11). Since each of the Area B risks is lower, this alternative is dominant. Respondents who do not comprehend the task and incorrectly answer that they prefer Area A are sent through a series of questions that explain the structure of the choice in more detail.

The performance with respect to the dominance question was quite good. Over four-fifths of the sample gave a correct response to the dominance questions on their initial attempt. After being given additional information, fewer than one percent of them gave an incorrect answer, and these respondents were excluded from the sample since they did not understand the interview task.

The program then proceeds with a series of pairwise comparisons in which the attributes are altered based on the previous responses until indifference is achieved. The computer program used tabular summaries, but for expositional purposes we will consider the abstract formulation of the trade-offs.

## A Model of State-Dependent Utilities

Consider the following model of state-dependent utilities. Let subscript a denote Area A and b denote Area B. Also, let U(CB) be the utility of a case of chronic bronchitis, U(D) equal the utility of an auto accident death, and U(H) equal the utility of being healthy (i.e., having neither CB nor an auto accident). To simplify this exposition, we assume that contracting CB and dying from an automobile accident are mutually exclusive events. Also, let $X_a$ denote the probability x/100,000 for Area A and $Y_a$ denote the probability y/100,000 for Area A, and let $x_b$ and $y_b$ be defined similarly. The survey continually modifies the choice pairs until subjects reached the situation where

(1)
$$X_aU(CB) + Y_aU(D) + (1 - X_a - Y_a)U(H)$$
$$= X_bU(CB) + Y_bU(D) + (1 - X_b - Y_b)U(H).$$

Our general objective is to establish the death risk equivalent of chronic bronchitis. If we assume for concreteness that $X_a > X_b$ and $Y_b > Y_a$ (no loss of generality), then

(2)
$$(X_a - X_b)U(CB) = (Y_b - Y_a)U(D) + (X_a - X_b + Y_a - Y_b)U(H),$$

or

(3)
$$U(CB) = \frac{Y_b-Y_a}{X_a-X_b}U(D) + (1 - \frac{Y_b-Y_a}{X_a-X_b})U(H) \quad .$$

If we define the rate of trade-off between CD and D as $t_1$, so that

(4)
$$t_1 = \frac{Y_b-Y_a}{X_a-X_b} \, ,$$

we obtain the result that

(5)
$$U(CB) = t_1U(D) + (1 - t_1)U(H) \quad .$$

The utility of CB cases has been transformed into an equivalent lottery on life with good health and death, for which we have a well-developed literature.

Survey Structure

Now consider the first set of paired comparison questions presented in Questionnaire A after the dominant choice question described above. In this case, respondents are given the choice between Area A with risks (75, 15) and Area B with risks (55, 19). Suppose that Area B is preferred in this example. Area B has the lower chronic bronchitis risk and higher auto accident risk; therefore, in subsequent questions the program raises the CB risk in the preferred Area B until indifference is achieved. If in the original choice the subject prefers Area A, in subsequent questions the program lowers the auto death risk in Area B until the point of indifference is reached.

Suppose that after considering a series of such comparisons the subject reaches indifference where he views the risk (75, 15) as being equivalent to (65, 19). Using equations 4 and 5 above, this would imply that

$$t_1 = \frac{19-15}{75-65} = 0.4$$

and

$$U(CB) = 0.4U(D) + 0.6U(H).$$

The second set of paired comparison questions in Questionnaire A focuses on the more traditional risk-dollar trade-off involving CB and cost of living. Area A has the same cost of living as the respondent's present residence, but Area B has a cost of living that is \$80 higher, yet poses a lower CB risk $X_b$. If in the initial question Area B is preferred, Area B's CB risk is increased until indifference is achieved. Similarly, if Area A is preferred, Area B's cost of living is reduced until reaching the point of indifference.

In the context of a state-dependent utility function with two arguments, health status and income, we have

$$X_aU(CB) + (1 - X_a)U(H) = X_bU(CB,-\$80) + (1-X_b)U(H,-\$80).$$

If utility functions are additively separable in money and health, then

$$X_aU(CB) + (1 - X_a)U(H) = X_bU(CB) + (1-X_b)U(H) + U(-\$80),$$

which simplifies to

$$(X_a - X_b)U(CB) = U(-80) + (X_a - X_b)U(H),$$

or

$$U(CB) = \frac{U(-\$80)}{(X_a-X_b)} + U(H).$$

If we assume that utility is linear in money (with a coefficient equal to one) in establishing our health valuation scale, then we have

$$U(CB) = -L + U(H),$$

i.e., CB is equivalent to being healthy and suffering a financial loss tantamount to L dollars, where

$$L = \frac{-\$80}{X_a - X_b} .$$

This procedure to establish a risk-dollar trade-off rate involves two assumptions regarding the structure of utility functions. First, we assume additive separability with respect to money and health. Second, we assume that the dollar magnitudes treated are sufficiently small that utility is approximately linear in money. Since even risk-averse utility functions meet this test for small monetary changes,[10] we selected our health-risk levels so that the dollar magnitudes involved be small.

The structure of Questionnaire B is similar to Questionnaire A except the certain $80 loss in terms of living costs has been replaced by a lottery on $2000 storm damage loss. In this case, respondents must specify the storm damage probability that establishes an equivalent CB-storm damage pair. If we assume that respondents are risk-neutral, then the storm damage loss can be replaced by its expected value. The possible advantage over the cost-of-living approach is that respondents may be able to make more meaningful comparisons of two different lotteries rather than having one attribute -- the dollar payoff -- being non-stochastic. As with the first set of questions in Questionnaire A, if the consumer prefers Area B in the initial question, the program leads the consumer to indifference by increasing the CB risk of Area B until indifference is achieved. Similarly, when the consumer initially prefers Area A, the program reduces the storm damage risk in Area B until reaching the point of indifference.

Questionnaire C repeats the first part of Questionnaire A, and these samples are pooled in the analysis below. The second set of questions addresses the more traditional death risk--dollar trade-off using auto deaths and cost-of-living trade-offs. The structure is similar to that of the second set of questions in Questionnaire A except that CB has been replaced by auto fatality risks so that respondents must reach the point that

$$U(D) = -L + U(H),$$

where

$$L + \frac{-80}{X_a - X_b} \quad ,$$

as before. This portion of the study provides a direct comparability test with the literature on market-based values of life. The fatality risk--dollar trade-offs will also be used in conjunction with the chronic bronchitis--fatality risk trade-offs to establish a chronic bronchitis--dollar trade-off rate.

Table 2 summarizes the structure of the 3 questionnaires described above.

## Sample Description

The interviews of the subjects were all done through an interactive computer program, thus avoiding problems of interviewer bias and promoting honest revelation of preferences. Response rates to sensitive questions, such as income level, were

Table 2

Summary of Survey Structure

Questionnaire A

| | Trade-Off | Units of Measurement | Procedure |
|---|---|---|---|
| 1. | Chronic bronchitis - auto deaths | Auto deaths per chronic bronchitis case | In the area with the higher auto accident risk, increase the bronchitis risk (to make that area less desirable) or reduce the auto accident risk (to make that area more desirable) until reaching indifference. |
| 2. | Chronic bronchitis - cost of living | Dollar value per 1/100,000 reduced risk of bronchitis | In the area with lower bronchitis risk, increase the bronchitis risk (to make that area less desirable) or decrease the cost of living (to make that area more desirable) until reaching indifference. |

Questionnaire B

| | Trade-Off | Units of Measurement | Procedure |
|---|---|---|---|
| 1. | Chronic bronchitis - storm damage | Reduced probability of $2000 storm damage that is equivalent to one bronchitis case prevented | In the area with the higher storm damage risk, increase the bronchitis risk (to make that area less desirable) or reduce the storm damage risk (to make that area more desirable) until reaching indifference. |

Table 2 (cont'd)

Summary of Survey Structure

Questionnaire C

| | | |
|---|---|---|
| 1. Chronic bronchitis - auto deaths<br>(Same as Questionnaire A - Part 1) | Auto deaths per chronic bronchitis case | In the area with the higher auto accident risk, increase the bronchitis risk (to make that area less desirable) or reduce the auto accident risk (to make that area more desirable) until reaching indifference. |
| 2. Auto accidents - cost of living | Dollar value per 1/100,000 reduced risk of an auto accident | In the area with lower auto accident risk, (to make that area less desirable) or decrease the cost of living (to make that are more desirable) until reaching indifference. |

much higher than those usually achieved with face-to-face interviews. In addition, subjects were not concerned with whether their responses impressed the interviewer. Use of a computer also made it possible to ask a sequence of questions to ascertain the appropriate marginal rates of substitution.

The sample was recruited for the study by a professional marketing firm at a mall intercept in Greensboro, North Carolina. This locale has a representative household mix and is used as a test marketing site for many national consumer brands. This firm and locale have been used successfully in two previous studies by the authors.[11] Use of such a consumer sample also yields more reliable responses to issues such as the valuation of property damage from storms than would a student sample or a sample from a city with an unrepresentative population, such as the college-oriented cities of Evanston, Illinois, or Chapel Hill, North Carolina.

Table 3 provides a glossary of the variables and the associated sample statistics. Questionnaires A and C had a similar mix of respondents, with a mean age in the low thirties, a even split between males and females, two years of college education, a 50 percent married rate, about 0.6 children under 8 years old, a household size of 2.7 - 2.8, and a household income in the mid-range of thirty to forty thousand dollars. Questionnaire B has a somewhat different mix because of the difference in the times at which the samples were recruited (e.g., week-end shoppers differ from day-time weekday shoppers). The Questionnaire B sample is about 10 years older, more likely

## Table 3

## Summary of Sample Characteristics

| | Mean and Std. Deviations | | |
|---|---|---|---|
| | Questionnaire | | |
| Demographic Variables | A | B | C |
| AGE, in year | 33.74<br>(12.42) | 43.47<br>(12.68) | 33.07<br>(11.66) |
| MALE, sex dummy variable | 0.50 | 0.42 | 0.51 |
| EDUCATION, years of schooling | 14.02<br>(2.23) | 14.32<br>(2.47) | 13.79<br>(2.66) |
| MARRIED, married dummy variable | 0.49<br>(0.50) | 0.79<br>(0.41) | 0.49<br>(0.50) |
| KIDS, number of children under 8 | 0.56<br>(1.00) | 0.83<br>(1.04) | 0.65<br>(1.07) |
| HOUSEHOLD, number of people in household | 2.71<br>(1.25) | 3.00<br>(1.16) | 2.80<br>(1.23) |
| INCOME, annual household income in dollars | 35,386.60<br>(19,009.95) | 45,367.65<br>(20,335.54) | 37,153.85<br>(21,333.80) |
| | 194 | 204 | 195 |

to be married, and with a household income about $10,000 greater. As the last row of Table 3 indicates, each of the three samples had about 200 respondents, with combined sample for the study of 593.

## 3. Risk-Risk Trade-Offs

Table 4 displays the means and standard deviations of the trade-off rates implied by the indifference points of the subject responses. To go beyond these summary statistics, consider first set of trade-offs between CB and auto accident deaths. For this analysis Questionnaires A-1 and C-1 are pooled since the questions are identical.

Establishing a death risk metric for CB enables respondents to think in risk terms, avoiding the comparability problems that might be encountered if monetary attributes were introduced. Similarly, for policy purposes EPA can establish a death risk equivalent and establish cost-effectiveness ratios in terms of the cost per statistical death prevented. As indicated in Viscusi (1986), this cost-effectiveness index will provide a comprehensive measure of the policy impact and also avoid the political sensitivities of placing dollar values on all health outcomes. Once a uniform health metric is established, one can then compare the cost per life equivalent saved with various value-of-life reference points and decide whether the policy should be pursued if one wishes to take a benefit-cost approach.

Unlike market-based studies of the value of life, the survey technique yields information on the entire distribution of the valuations. Table 5 reports the deciles of the distribution for

## Table 4

## Rates of Trade-off Implied by Indifference Points

| | Means and Std. Deviations | | |
|---|---|---|---|
| Part B | Questionnaire | | |
| Trade-off Rates | A | B | C |
| CB-Auto (A-1 & C-1), auto deaths per CB case | 0.68<br>(0.82) | - | 0.70<br>(0.95) |
| CB-Cost of Living (A-2), dollar value per 1/100,000 CB risk | 8.83<br>(12.50) | - | - |
| CB-Storm Damage (B-1), number of $2,000 storms equal to one CB case | - | 852.60<br>(1064.20) | - |
| Auto-Cost of Living (C-2), - dollar value per 1/100,000 reduced auto accident risk | - | - | 81.84<br>(168.54) |
| Sample Size | 194 | 204 | 195 |

Table 5

Distribution of Chronic Bronchitis --

Auto Death Trade-Offs

| Decile | Auto Death Equivalents per Chronic Bronchitis Case |
|---|---|
| .10 | 0.12 |
| .20 | 0.20 |
| .30 | 0.23 |
| .40 | 0.27 |
| .50 (median) | 0.32 |
| .60 | 0.40 |
| .70 | 0.80 |
| .80 | 1.00 |
| .90 | 1.33 |
| 1.00 | 4.00 |
| Mean | 0.68 |
| (st. error of mean) | (0.06) |

respondents who gave consistent answers that converged to a particular trade-off value. Subjects whose responses indicated that they did not fully comprehend the valuation task were excluded from our sample.

Specifically, we excluded subjects who failed one of the following consistency checks:

1) they started the series of paired comparison questions by preferring one area, say Area A, and as Area B was made more desirable in subsequent comparisons they continued to prefer Area A, even on the last question of the series in which Area B dominated Area A on both attributes;
2) like inconsistency #1, they continued to prefer Area A in each comparison until the last one in which Area B dominated Area A in both attributes, yet on this last question they indicated indifference between Area A and Area B;
3) they indicated preference for one area, say Area A, on the first and all subsequent questions in the series (including the last one in which Area B dominated Area A), then when confronted with this inconsistency and asked to repeat the series of questions chose Area B in the first question (despite have selected Area A the first time they were given this question);
4) they indicated preference for one area, say Area A, on all questions in the series except the last one in the series (in which Area B dominated Area A) but including the next-to-last question (for which Area B easily dominated Area A on one attribute and Area A just barely dominated Area B on the

other attribute), thus making it impossible to interpolate between the trade-offs implied by the last two questions to obtain an indifference point (because the last question yields no rate of trade-off); or

5) they expressed indifference between all pairs of areas in the series of questions, despite wide variation in their attributes.

Individuals who failed one of these inconsistency checks either did not understand the choice task, were not responding honestly, attached no value to one of the two attributes, or have non-monotonic preferences for one of the attributes. We assume that neither of the last two preferences attributes are possessed by any subjects, thus implying that answers which fail any of the five inconsistency checks do not represent the subjects' true preferences.

The requirement that the response pattern to the series of paired comparisons be internally consistent will lead to more meaningful estimates than if no such checks were imposed. About two-thirds of the sample converged to an indifference situation and had consistent responses, where this percentage was similar across all questionnaires.[11] These consistency checks distinguish our approach from the usual contingent valuation method in which respondents' answers are taken at face value without such formal tests of whether the subjects understood the valuation task and displayed consistent choices.

In evaluating the distribution in Table 5, first consider the respondent at the tenth percentile. This person viewed a chronic bronchitis probability as being just as severe as a risk of an auto accident that was 0.12 as great. Thus, this individual would view a chronic bronchitis risk of 100/100,000 risk of 100/100,000 per year as being equivalent to the annual chance of being involved in an auto accident of 12/100,000.

Now examine the respondent at the other end of the distribution. This individual views a chronic bronchitis risk as being four times as severe as a risk of death, so that a 100/100,000 risk of CB would be viewed as comparable to a 400/100,000 risk of death. He or she gave consistent responses to the questions, but opted for the choice reflecting the highest CB valuation.

Many studies in the survey valuation literature exclude the tails of the distribution since they are tainted by extreme respondents such as this. Rather than discard such information altogether, we report the entire distribution, recognizing that the top and bottom deciles may be affected by a lack of complete understanding of the interview task. The reported distributions enable readers to assess how important outliers are within the context of the study and by focusing primarily on the median responses rather than the mean we avoid the distortion of our results by these outliers.

The response pattern in which CB was more highly valued than auto death risks was exhibited by the top two deciles for each questionnaire's response distribution. Such a pattern is not necessarily implausible. In addition to possibly misunderstanding the interview task, two explanations can be offered. First, individuals might legitimately believe that such a severe chronic illness is a worse outcome than death. The health outcome described in Table 1 is quite serious and will have substantial duration. Their normal activities would be curtailed, medical interventions including hospitalization and possible reliance on a portable oxygen tank would accompany severe cases of CB, other illnesses would be more likely, and they would experience periods of depression.

The second possible explanation is that the respondents were establishing equivalencies between different average risks in an area rather than different risks to themselves. The CB risk was characterized as an involuntary risk not under their control except for smoking, whereas the auto accident risk differs depending on one's driving habits and skills. Other studies suggest that individuals may have overly optimistic assessments of risks influenced by their actions, such as auto death risks, as discussed in Viscusi and Magat (1987). If this were the case, the perceived person-specific risk would be below the stated risk, causing an upward bias in the results in Table 5.

The median CB valuation is equivalent to 0.32 auto deaths. Because of the skewed nature of the responses, the mean value of 0.68 is more than double the median response. Regression analysis of the CB-auto death trade-off rates indicate no significant variation across subjects with respect to either demographic factors such as age, income, and education, or personal characteristics such as smoking habits. This result is neither surprising nor disturbing. Most individual attributes, such as household income, should affect the CB valuation and the value of life similarly, and thus be unrelated to variation in the CB--auto death trade-off rates across subjects. Because there are no systematic differences among individuals in their risk-risk trade-offs, we can aggregate them into meaningful summary measures such as medians and means without the risk of drawing misleading conclusions from an unrepresentative sample.

The general implications of these results is a follows. Most, but not all, people regard the risk of chronic bronchitis as a less severe outcome than the risk of death. However, the prospect of a sustained chronic illness is viewed as a very severe outcome. Based on the median responses, the death risk equivalent of CB is 0.32, and based on the mean response it is 0.68. The general order of magnitude of both the median and the mean is the same and is just below that of fatalities. As will be indicated in Section 5, these statistics can be transformed into dollar valuation equivalents using established value-of-life statistics.

## 4. Risk-Dollar Valuations of Chronic Bronchitis

The second approach that we employed to value chronic bronchitis was to establish risk-dollar trade-offs. The two approaches used were to establish the chronic bronchitis risk equivalent of a higher cost of living and to determine the relationship between chronic bronchitis risks and storm damage risks.

Consider first the cost-of-living results in Table 6. The first column of Table 6 lists the decile of the distribution.

## Table 6

## Distribution of Chronic Bronchitis - Cost of Living Trade-Offs

| Decile | Trade-Off Levels<br>Dollar Value per 1/100,000 Reduced Risk of Chronic Bronchitis (A-2) | Implicit Dollar Value per Case of Chronic Bronchitis |
|---|---|---|
| .10 | 1.50 | $150,000 |
| .20 | 3.00 | $300,000 |
| .30 | 3.50 | $350,000 |
| .40 | 4.00 | $400,000 |
| .50 (median) | 4.57 | $457,000 |
| .60 | 5.33 | $533,000 |
| .70 | 6.40 | $640,000 |
| .80 | 8.00 | $800,000 |
| .90 | 20.00 | $2,000,000 |
| 1.0 | 80.00 | $8,000,000 |
| Mean | 8.83 | $883,000 |
| (st. error of mean) | (1.14) | ($114,000) |

Column two presents the increased dollar value in the annual cost of living that the respondent was willing to incur per 1/100,000 reduction in the annual probability of chronic bronchitis. If we multiply the results in column 2 by 100,000, we obtain the implicit dollar value per statistical case of chronic bronchitis.

As in the case of the risk-risk results, the response pattern is skewed so that the upper tail of the responses generates a mean valuation estimate in excess of the median. The results here indicate the average dollar value of chronic bronchitis is $883,000, with an associated standard error of $114,000. The $457,000 median of the distribution is just over half of the mean. Each of these values is below the usual estimates of the implicit value of life, which are reviewed in Viscusi (1986). These results follow the expected pattern, given the CB--auto death risk trade-off results reported above.

As in the case of the risk-risk trade-offs, the upper bound of the chronic bronchitis valuation estimates exceeds most estimates of the value of a fatality, as $8 million exceeds some but not all estimates of the value of life. More precise comparisons of all of the results using a dollar metric will be undertaken in Section 5.

The second set of CB risk-dollar trade-offs, which is reported in Table 7, uses storm damage risks as the dollar counterpart so that respondents must compare monetary lotteries and health status lotteries rather than certain monetary (cost of living) differences with health status lotteries. The first column of results gives the value of y for which a storm causing

## Table 7

## Distribution of Chronic Bronchitis -- Storm Damage Trade-offs

| Decile | Equivalent $2000 Damage Probability (x100,000) | Implicit Dollar Value per Case of Chronic Bronchitis |
|---|---|---|
| .10 | 175.00 | $350,000 |
| .20 | 228.57 | $457,140 |
| .30 | 266.67 | $533,340 |
| .40 | 266.67 | $533,340 |
| .50 (median) | 400.00 | $800,000 |
| .60 | 533.33 | $1,066,660 |
| .70 | 800.00 | $1,600,000 |
| .80 | 1,333.33 | $2,666,660 |
| .90 | 2,000.00 | $4,000,000 |
| 1.0 | 4,000.00 | $8,000,000 |
| Mean | 852.60 | $1,705,200 |
| (st. error of mean) | (91.93) | ($183,860) |

damage of $2000 with a probability of y/100,000 is equivalent to a chronic bronchitis probability of 1/100,000. A more meaningful metric is the expected storm damage that is equivalent to each CB case. This figure is obtained by multiplying the first column of results by the $2000 damage per storm damage event. The second column of results gives the dollar value per statistical case of chronic bronchitis, where these dollar values have been obtained using the storm damage costs.

A comparison of the distributions of implied CB valuations in Tables 6 and 7 suggests that the subjects may have found the storm damage lottery comparison to have been more difficult to make than the comparison with a non-probabilistic cost-of-living increase. The distribution derived from the storm damage lottery comparison stochastically dominates the distribution from the cost-of-living comparison, with both its median and mean almost double that of the cost-of-living distribution. Based on a comparison with the dollar values of avoiding automobile accident fatalities reported in next section, the CB avoidance values derived from the storm damage lottery questions appear to be somewhat high. Further, the standard error of the mean is about 50 percent higher for the distribution derived from the storm damage distribution than for the cost-of-lived based distribution of CB values. In any event, these results do not suggest that expressing dollar trade-offs in probabilistic form, as in the storm damage lottery, aids people in making risk-dollar trade-offs, which was our original hypothesis.

### 5. Trade-Offs Between Auto Deaths and Cost-of Living

A useful check on the survey methodology is to ascertain the implicit value of life using a direct fatality risk-dollar trade-off. This is done using automobile accident risks and cost of living in Questionnaire C-2, and the results of this exploration are reported in Table 8.

The median response of $2,286,000 is quite reasonable in view of the similar (in 1987 dollars) market-based estimate by Blomquist (1979), but the mean value of $8,184,000 seems rather large. The high mean estimate was generated by a portion of the sample with value of life estimates as high as $80,000,000. Such implausibly large estimates can occur because of the difficulty of the comparison task. Respondents are being asked to establish an equivalence between some annual chance of chronic bronchitis x/100,000 that is equivalent to an $80 cost-of-living increase. This is a difficult comparison to make. In contrast, the risk-risk questions focused on chronic bronchitis--auto accident risk comparisons of x/100,000 and y/100,000, where most respondents did not believe that the severity of outcomes differed by more than an order of magnitude.

The implicit dollar value of CB can be obtained by chaining the responses to questionnaire part C-1, which gives the CB-auto death trade-off, and part C-2, which gives the auto death--dollars trade-off. These results appear in Table 9. The median dollar value of each chronic bronchitis case is $800,000. The mean is much greater because there is one outlier with a $320 million value. This individual expressed extreme responses on

## Table 8

## Distribution of Auto Accident -- Cost of Living Trade-Offs

| Decile | Dollar Value per 1/100,000 Reduced Risk of an Accident | Implicit Dollar Value of an Accident |
|---|---|---|
| .10 | 10.00 | $1,000,000 |
| .20 | 17.50 | $1,750,000 |
| .30 | 17.50 | $1,750,000 |
| .40 | 20.00 | $2,000,000 |
| .50 (median) | 22.86 | $2,286,000 |
| .60 | 26.67 | $2,667,000 |
| .70 | 40.00 | $4,000,000 |
| .80 | 80.00 | $8,000,000 |
| .90 | 177.78 | $17,778,000 |
| 1.0 | 800.00 | $80,000,000 |
| Mean | 81.84 | $8,184,000 |
| (st. error of mean) | (14.40) | ($1,440,000) |

## Table 9

## Implicit Valuation of Chronic Bronchitis Implied by CB--Auto Death and Auto Death -- Cost of Living Trade-offs

| Fractiles | Questionnaire C<br>Inferred CB Value |
|---|---|
| .10 | $200,000 |
| .20 | $350,000 |
| .30 | $522,449 |
| .40 | $646,154 |
| .50 | $800,000 |
| .60 | $1,066,667 |
| .70 | $2,133,333 |
| .80 | $3,555,556 |
| .90 | $12,800,000 |
| .99 | $71,111,111 |
| 1.00 | $320,000,000 |
| Mean<br>(Std. Error of Mean) | $6,962,364<br>($2,977,373) |
| | (N = 112) |

each component part, valuing each CB case at four times the amount of each death and having an implicit value of an auto fatality of $80 million. In each case, these were the highest values in the sample and the highest permitted by the program, which indicates that this individual probably did not understand the valuation task.

As instructive summary of the results is provided in Table 10. For the results creating CB/auto death risk equivalents, the numbers have been transformed into implicit value-of-life terms using three different reference points: a $2 million value of life; a $3 million value of life; and a $5 million value of life. The $2 million figure is comparable to the median auto death risk valuation within the survey so that this estimate provides an internal comparison of the results. The $3 million figure is included since the recent estimates by Moore and Viscusi (1988) indicate that the labor market value of life is in the $2-$3 million range using BLS data, and this was the "best estimate" of the value of life in earlier work by Viscusi (1983). The $5 million reference point is the value of life figure obtained using new NIOSH data on job fatality risks, which Moore and Viscusi (1988) view to be superior to the BLS data.

The pattern displayed by the results is fairly similar. In each case mean valuations are at least double the value of the median. Although one would not expect symmetry in a distribution truncated at zero, the very high end responses observed appear to be due to response errors.

Table 10

Summary of Risk-Dollar Equivalents

| | Direct Valuation Estimate | CB Estimate Using $2 Million Value of Life | CB Dollar Estimate Using $3 Million Value of Life | CB Dollar Estimate Using $5 Million Value of Life |
|---|---|---|---|---|
| CB/Auto Fatality: | | | | |
| A-1 & C-1 (Median) | -- | $640,000 | $960,000 | $1,600,000 |
| A-1 & C-1 (Mean) | -- | $1,360,000 | $2,040,000 | $3,400,000 |
| CB/Cost of Living: | | | | |
| A-2 (Median) | $457,000 | -- | -- | -- |
| A-2 (Mean) | $883,000 | -- | -- | -- |
| CB/Storm Damage: | | | | |
| B-1 (Median) | $800,000 | -- | -- | -- |
| B-1 (Mean) | $1,705,200 | -- | -- | -- |
| CB/Dollars (Derived from CB/Auto Fatality and Auto/Cost of Living): | | | | |
| C-1 & C-2 | $800,000 | -- | -- | -- |
| C-1 & C-2 | $6,962,364 | -- | -- | -- |
| Auto/Cost of Living: | | | | |
| C-2 (Median) | $2,286,000 | -- | -- | -- |
| C-2 (Mean) | $8,184,000 | -- | -- | -- |

The most clearcut divergence from plausible patterns is the mean value of life of $8,184,000 for the auto death/cost-of-living trade-off. Whereas the mean CB/auto values were roughly double the median, the mean auto/cost of living values were almost four times the size of the median, indicating a much more skewed distribution. As noted in the discussion of Table 8, this mean value was influenced in part by individuals with implied values of life as high as $80 million. These outliers suggest that for some people making meaningful trade-offs involving small cost-of-living differences and low risks of auto accident fatalities is a task they cannot handle effectively.

The valuation of chronic morbidity across the difference questionnaire approaches is quite similar for the case in which we use a $2 million value of life figure to transform the death equivalent statistics into meaningful dollar estimates. The median value for the CB/auto death risk trade-offs is $640,000, as compared with a median value of $457,000 for the CB/cost of living trade-off and a median value of $800,000 for the CB/storm damage results. These results are similar to the $800,000 median CB value that was obtained by chaining the CB/auto and auto/cost of living responses. Even with a higher value of life of $3 million, the CB/auto median of $960,000 is not out of line with the CB/cost of living and CB/storm damage results.

Once we move to the case where a $5 million value of life is used, the median dollar valuation of each CB case prevented is greatly increased to the $1,600,000. If EPA were to rely on, for example, the CB/cost of living results to value CB and then use a value of life of $5 million without also using an appropriately adjusted CB value, this procedure could potentially understate the value of the CB cases prevented by a factor of three. By converting all outcomes to a health risk equivalence scale using a death risk metric, EPA avoids any distortion in the mix of targeted illnesses that might otherwise occur if the value of life number selected was incorrect.

## 6. Conclusion

Although market evidence remains our most reliable guideline for assessing the shape of individual preferences, such evidence is unavailable for many outcomes that are either not traded explicitly in markets or traded implicitly but in a market for which available data are not rich enough to identify the pertinent trade-off rates. Analysis of risk-risk and risk-dollar trade-offs using various types of simulated market choices provides a useful mechanism for establishing such values.

This study has developed a methodology for deriving morbidity valuation estimates based on the trade-off with another well-known risk, rather than forcing individuals to express trade-off rates between morbidity rate reductions and dollars, a task which is unfamiliar to most people. We presented several conceptual reasons why consumers should be able to more accurately convey risk-risk trade-offs than risk-dollar trade-offs, and the application of our methodology to the valuation of reductions in the risk of chronic bronchitis indicate that most individuals can make risk-risk trade-offs, even with a disease as complicated and unfamiliar to healthy people as chronic bronchitis.

Although for the purpose of cost-effectiveness analysis there is no need to measure risk reduction value in terms of dollars, when we translated our risk-risk estimates into risk-dollar estimates using either survey results on auto accident risk reduction values or published value-of-life estimates, the distributions compared favorably, thus providing additional confidence in the reasonableness of the results derived from our methodology. While this study applied the approach to the valuation of only two risks, that of chronic bronchitis and an auto accident fatalities, the favorable results suggest that the methodology may be more widely applicable to other morbidity risks, such as various forms of cancer.

## Footnotes

[1] See Viscusi (1986) for a review of the market trade-off literature.

[2] See analysis by Blomquist (1979) for an inventive use of seatbelt usage data to infer a value of life.

[3] Survey studies of various health and environmental risks include the seminal work by Acton (1973) as well as more recent studies often grouped under the designation "contingent valuation." These recent analyses include: Brookshire, Thayer, Schulze, and d'Arge (1982); Cummings, Brookshire, and Schulze (1986); Fischhoff and Furby (1988); Gerking, de Haan, and Schulze (1988); Smith and Desvousges (1987); Viscusi and Magat (1987); Viscusi, Magat, and Forrest (1988); and Viscusi, Magat, and Huber (1987); and Fisher, Chestnut, and Violette (1989).

[4] In designing our survey, we used software from Sawtooth Software, Inc.

[5] For an important recent study of the valuation of health risks rather than mortality, see Berger et al. (1987).

[6] For example, see Viscusi, Magat and Huber (1987), pages 477-478.

[7] See Petty (1985) for a discussion of the distinction between chronic bronchitis, the related disease emphysema, and the broader disease category called chronic obstructive pulmonary disease. The authors selected the type of chronic bronchitis described in Table 1 after consulting closely with two lung specialists at Duke University Medical Center and visiting the Medical Center rehabilitation program for patients with severe lung diseases.

[8] At the end of the interview, subjects were carefully debriefed about this use of average rather than person-specific risks.

[9] Our past studies suggest that presenting the risk in terms of the number of cases for a large base population is more comprehensible than giving risk levels such as 0.00075.

[10] See Arrow (1971).

[11] See Viscusi and Magat (1987) and Viscusi, Magat, and Huber (1987).

[12] Probit analysis was used to identify personal characteristics that explain the division of subjects between those giving consistent and inconsistent responses. The only two significant variables in the equation are AGE and SMOKER, with older respondents less likely to give consistent responses and smokers more likely to respond consistently. These results may reflect the difficulty that older subjects have with the new interview technology (computers) and the greater thought that smokers have given to the implications of chronic bronchitis.

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# THE SOCIAL COSTS OF CHRONIC HEART AND LUNG DISEASE

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## INTRODUCTION

To value a program that reduces the probability of contracting a chronic disease, one would like to know what a person who does not have the disease would be willing to pay to reduce his probability of getting it. The sum across individuals of these willingnesses to pay, plus the expected costs of the disease that are not borne by these individuals, comprise the theoretically correct measure of social benefits from reducing incidence of the disease.

In this paper we measure the medical costs and lost productivity associated with various chronic heart and lung diseases. Our justification for focusing on these components of the social cost of illness is that medical costs and lost earnings are often not borne by individuals themselves and, hence, are unlikely to be reflected in willingness to pay figures. Therefore, they must be added to willingness to pay estimates to compute the total benefits of reducing the incidence of a disease.

### Effects on Earnings

Our estimates of the effects of chronic illness on labor force participation and on earnings differ in two respects from those available in the literature (Bartel and Taubman, 1979; Salkever, 1985). First, our dataset--the Social Security Survey of Disabled and Non-Disabled Adults--allows us to distinguish the effects of individual diseases (e.g., emphysema, chronic bronchitis) rather than disease categories (chronic respiratory illness).[1] As one might expect, there is significant variation in the effects of individual diseases within broader categories: Emphysema, for example, has a large negative effect on earnings whereas chronic bronchitis does not. Hypertension has no significant effects on probability of participation or on earnings, whereas a heart attack occurring between 45 and 54 reduces both.

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[1] The diseases studied are: allergies, asthma, chronic bronchitis, emphysema, other chronic lung disease, arteriosclerosis, heart attack, hypertension, other chronic heart disease and stroke.

Second, we examine how the effect of each disease varies with age of onset and duration. It is generally believed (Bartel and Taubman, 1979) that, other things equal, a person is more likely to participate in the labor force at any age the earlier in life he contracts a chronic disease. The argument is that the benefits of making adjustments to the disease (retraining, changing occupations) are larger the earlier in life the disease begins. Thus, the earlier the age of onset the more likely it is that adjustments will be made. It is not, however, clear that the human capital argument applies to the diseases examined here, most of which are contracted later in life. Since one seldom witnesses changes in occupation after age 45 it is unlikely that small variations in age of onset matter after this age. Indeed, age of onset may have a positive effect on participation if a disease is more serious when contracted at an earlier age.

It is also of interest to see how the duration of a disease alters labor market behavior. For two persons who contracted emphysema at age 45, are effects on earnings greater for a person currently 50 or for a person currently 60? Holding age of onset constant, this is equivalent to asking whether the disease has a greater effect on participation and earnings when one has had the disease for five years or for fifteen years. One might hypothesize that the longer one has had a disease the longer he has had to adjust to it; hence, labor market effects should diminish with duration. On the other hand, for progressive diseases, e.g., emphysema, the longer one has had the disease the more serious it is likely to be.

We find that the tendency of chronic disease to reduce labor force participation and earnings does not increase with age of onset. Indeed, for emphysema, heart attack, arteriosclerosis and stroke, an age of onset between 45 and 54 significantly reduces the probability of working at all future ages, but an age of onset between 55 and 65 does not. It might seem that this result occurs because people who contract a disease earlier will, on average, have had it for a longer time than persons who contracted it later in life. For emphysema this appears to be true. When duration is held constant, it is having the disease for 6 or more years that affects labor market behavior rather than contracting it at age 45. For heart attack, arteriosclerosis and stroke, however, the duration of the disease, holding age of onset constant, has no effect on participation.

## Medical Costs

Our estimates of medical costs, which come from the National Medical Care Expenditure Survey (NMCES), have two advantages over existing estimates of medical expenditures (National Heart, Lung and Blood Institute, 1982; Hartunian et al., 1981). The National Heart, Lung and Blood Institute allocates aggregate costs, such as hospital costs and doctor costs to diseases based solely on a disease's proportion of total activities, e.g., hospital days and total doctor visits, respectively. This approach has two shortcomings: (1) it assumes that the average cost of, say, a hospital day or doctor's visit is the same for all diseases, and (2) it does not allow one to examine the distribution of medical costs per person. An alternative "engineering" approach is to multiply the number of hospital days or doctor visits attributable to a condition by the typical price for a hospital day or typical price for a doctor visit for that condition (see e.g., Freeman (1976)). This approach circumvents the first objection raised above but not the second.

By using individual data on medical costs, collected over a one-year period for over 40,000 persons, we are able to examine the distribution of medical costs per person by disease. Our most interesting results pertain to the size distribution of medical costs. For the five diseases whose medical costs we study--bronchitis, emphysema, hypertension, ischemic heart disease and non-specific heart disease--the distribution of annual costs per person is highly skewed. For emphysema, ischemic heart disease and non-specific heart disease median expenditures are less than one-tenth of mean expenditures. For bronchitis and hypertension median expenditures are about one-fourth of mean expenditures.

Because NMCES contains information on source of payment, it is also possible to see to what extent individuals and their families bear the medical costs of these diseases. For emphysema, ischemic heart disease and non-specific heart disease only about 10% of aggregate medical costs are borne by patients' families. The percentages are somewhat higher for bronchitis (34%) and hypertension (23%). The percent of cost borne by the patient's family differs, however, by size of cost. As noted above, the majority of persons with the diseases studied here incur small annual medical expenses. Averaging across individuals, the fraction of medical costs paid for by one's family is 2/3 for hypertension and bronchitis and half for emphysema, ischemic heart disease and non-specific heart disease. This implies that, on average, individuals (or their families) pay a higher fraction of small medical expenditures than of large ones.

## THE EFFECT OF CHRONIC ILLNESS ON LABOR FORCE PARTICIPATION AND EARNINGS

### The Model

In modelling the effects of various diseases on earnings it is standard practice (Bartel and Taubman, 1979; Mitchell and Butler, 1986) to distinguish the effects of each disease on participation from its effects on earnings given that one participates. Debilitating diseases such as emphysema and stroke may force a person to drop out of the labor force because he is physically unable to work, or may reduce earnings to the point where they fall below the reservation wage. If a person continues working he may curtail hours (if free to do so) or suffer a drop in pay because he changes jobs or because his productivity falls. This implies a drop in earnings, conditional on working.

The decision to participate, and earnings, conditional on participation, constitute a two-equation system. The individual participates if the decision function, $I_t$, is non-zero. Earnings, $Y_t$, are observed only if the individual participates.

$$I_t = Z_t\delta - e_t \qquad \text{Participation decision} \qquad (1)$$

Participate if $I_t \geq 0$,

$$Y_t = X_t\beta + u_t \qquad \text{Earnings in labor market} \qquad (2)$$

$Y_t$ observed if $I_t \geq 0$

$Y_t$ not observed if $I_t < 0$.

Equation (1) can be viewed as a reduced-form equation that results from comparing the utility received from income and leisure, conditional on working, with the utility received from income and leisure given that the individual does not work. If income and leisure in each state are replaced by their exogenous determinants, one obtains equation (1).[2]

Because earnings in (2) are observed only for working persons, estimation of (2) involves a classic selectivity problem: persons for whom earnings data are available are in the lower tail of the error distribution in equation (1). As long as the errors in equations (1) and (2) are correlated, applying least squares to (2) results in inconsistent parameter estimates since $E(u_t | Z_t \delta \geq e_t) \neq 0$.

To obtain consistent estimates of this system we follow the two-stage approach outlined by Lee (1983) [see also Maddala (1983)]. We assume that the error term in the participation equation has a logistic distribution $F(e_t) = 1/[1+\exp(-Z_t \delta)]$, and estimate a logit model of labor force participation. The error term $e_t$ can be transformed to an error term $e_t^*$ with a standard normal distribution,

$$e_t^* = J(e_t) = \Phi^{-1}(F(e_t)),$$

where $\Phi^{-1}$ is the inverse of the standard normal distribution function. Assuming that $e_t^*$ and $u_t$ are bivariate normally distributed with correlation coefficient $\rho$ and $V(u_t) = \sigma^2$, expected earnings are a linear function of $X$ plus a term $\phi/F$ that represents the density of $e_t^*$ conditional on working,

$$E(X_t \beta + u_t | e_t \leq Z_t \delta) = X_t \beta + \sigma\rho\phi[J(Z_t \delta)]/F(Z_t \delta) + v_t. \qquad (3)$$

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[2] This implies that all variables entering (2) should enter (1).

Applying OLS to (3) yields consistent estimates of the parameters $\beta$ and $\sigma\rho$.[3]

## The Data

### The Sample

The data used to estimate our model come from the 1978 Social Security Survey of Disability and Work (U.S. Department of Health and Human Services, Social Security Administration, 1981). The survey, which was designed to examine issues relating to eligibility for disability benefits and the effects of disabilities on labor force participation, consists of two samples, a stratified random sample of 6,853 persons from the 1976 Health Interview Survey, and a sample of 4,886 persons from the population of recipients of Social Security Disability Insurance who were declared eligible for benefits no earlier than 5 years before the survey. Our sample consists of 2,218 men between the ages of 18 and 65 from the Health Interview Survey portion of the Social Security Survey.[4]

### Earnings Equation

To avoid transitory fluctuations during the survey week, earnings are measured as wages and salaries received from all jobs during 1977. (All earnings are measured in 1977 dollars.) The independent variables entering the earnings equation, $X_t$, are listed in Table 1. Earnings are assumed to depend on education (measured by a series of dummy variables), experience (proxied by a series of age dummies), experience squared, marital status, family size, race, locational dummies and the health variables described below and in Table 2.

### Labor Force Participation Equation

As with earnings, participation is defined based on behavior throughout the 1977 calendar year. An individual is considered to have been in the labor force if he worked 30 or more weeks during the 1977. Men who did not work at all during 1977 are classified as not participating in the labor force. Men working between one and 29 weeks were eliminated from the sample on the grounds that these persons were either students or changed labor force status.

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[3] The two-stage estimation procedure, including asymptotic standard errors (Maddala, 1983), was programmed by the authors using the SAS matrix language.

[4] There are a total of 2,626 men between 18 and 65 in the HIS portion of the Social Security survey. 408 of them were eliminated because they appeared to change labor force status during 1977, the year for which participation and earnings were measured.

Since the decision to participate in the labor force is made by comparing the utility of income and leisure when in the labor force with income and leisure when out of the labor force, the variables in $Z_t$ should include all those entering the earnings equation, plus variables that would affect income conditional on not participating, and variables that would affect the utility of leisure time. The only such variables available in the survey that are not included in $X_t$ are (1) whether the individual is aware of Social Security disability benefits and (2) whether the individual is a veteran, both of which might affect income received if the individual did not participate. A third variable included in $Z_t$ to capture motives for working is the size of the respondent's debt.

Health Variables. The survey contains two types of information about chronic illness. Respondents were asked whether they had ever been diagnosed by a doctor as having any one of the 35 chronic diseases listed in Table 2, as well as when the disease first began to bother them (age of onset). They were also asked whether they were functionally limited by any of the diseases. Functional limitation questions include whether the respondent had difficulty walking, climbing stairs, lifting heavy objects, etc. Respondents were also asked whether they experienced symptoms such as pain, fatigue, swelling and shortness of breath.

In both the earnings and participation equations the severity of chronic disease is measured by dummy variables that indicate the presence of a chronic condition. Measures of functional limitation, while possibly useful as indicators of the severity of disease, are not associated with specific diseases and, hence, cannot be used to measure the severity of individual diseases.[5]

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[5] In addition to collecting these measures of functional limitation, the survey also asks respondents if they "have a disability that limits the type or amount of work [they] can do?" This variable, which is included in addition to the chronic disease dummies in Mitchell and Butler's (1986) analysis of the labor market effects of arthritis, was excluded from our analysis for two reasons. First, the answer to this question is not an exogenous measure of health but reflects the

Footnote 5 continued on next page

In measuring the effect of particular diseases on participation and on earnings we would like to distinguish effects by age of onset and by duration of the disease. The extent to which this is possible depends on the disease studied. Table 3 gives the distribution of age of onset for persons in our sample for each of the 10 respiratory and circulatory diseases studied. In our sample few cases of emphysema, arteriosclerosis, or stroke occur before age 45. For this reason these diseases are represented by only two age of onset dummies indicating that the disease was contracted between the ages of 45 and 54 or between the ages of 55 and 65.

Chronic bronchitis and other chronic lung disease occur earlier in life than emphysema; however, the small numbers of persons in our sample with these conditions restrict us to only two age of onset categories for each disease: before age 45 and after age 45. Allergies, asthma, heart attack, hypertension, and other chronic heart disease occur frequently enough and early enough in life that we can distinguish between 3 and 5 age of onset categories for each disease, as indicated in Table 2.

We have attempted to distinguish between duration of disease and age of onset only for those diseases that appeared to have a significant effect on labor force participation when age of onset alone was measured.[6] These included emphysema, arteriosclerosis, heart attack, stroke and other heart disease. Each disease was significant only when age of onset was 45 or older. The fact that these diseases occur later in life, together with a maximum sample age of 65, means that we can distinguish only two duration categories: persons who have had the disease 0-5 years and persons who have had the disease 5-10 years.[7]

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Footnote 5 continued from previous page
decision to stop/continue working. Second, the variable may capture effects of multiple diseases that we wish to capture using disease-specific dummies.

[6] Throughout the paper "statistically significant" means significant at the 5% level, one-tailed test.

[7] Chronic bronchitis beginning between ages 25 and 44 significantly decreased the probability of labor force participation; however, there were too few persons who had had chronic bronchitis for more than 10 years to permit using additional duration dummies for this disease.

## Results

Labor Force Participation. The more serious respiratory and circulatory diseases examined--chronic bronchitis and emphysema; arteriosclerosis, heart attack, stroke and other heart disease--significantly reduce the probability that a man participates in the labor force, other things equal. Table 4 presents coefficients obtained from the logistic participation equation for the respiratory and circulatory disease variables listed in Table 2. [The coefficients of other variables in the participation equation appear in the appendix to this paper.] The table indicates that the less serious diseases--allergies, asthma, other chronic lung disease and hypertension--have no significant effects on participation. To calculate the effect of each disease on probability of participation its coefficient must be multiplied by $P(1-P)$, where $P$ is the probability of participation. Since $P = 0.670$ for our sample, the coefficients in Table 4 imply that contracting emphysema between ages 45 and 54 reduces the probability of participating in the labor force by an average of 23.3 percentage points. Arteriosclerosis reduces probability of participation by 15.6 percent, while having a stroke between 45 and 54 reduces subsequent probability of participation by 57.3 percent.

What is somewhat surprising is the effect of age of onset on participation. For emphysema, arteriosclerosis, heart attack and stroke, an age of onset between 45 and 54 significantly reduces probability of working at all future ages, but an age of onset between 55 and 65 does not. Such a result runs counter to the standard argument that, the earlier the onset of a disability, the more likely it is that the individual will adjust to it by retraining and/or switching jobs. One reason that the standard argument may not apply is that, for the diseases studied here, a diagnosis at age 45 may indicate a more severe case of the disease than a diagnosis at age 60 (a heart attack at age 45 is often more devastating than a heart attack at age 60).

A second possibility is that for progressive diseases such as emphysema and arteriosclerosis, persons who contract the disease earlier will, on average, have had it for a longer time than persons who contract it later in life. To the extent that severity increases with the duration of the disease, persons who have had the disease longer will be less likely to work.[8] The results in Table 4 may thus be due to the fact that age of onset is directly correlated with the number of years the individual has been bothered by the disease.

To test this hypothesis the age of onset categories in Table 2 were subdivided to distinguish duration of disease from age of onset. Persons with an age of onset between 45 and 54 were divided into two categories: those who had had the disease for 0-5 years and those who had had the disease for 6-10 years. For persons with an age of onset between 55 and 65 only the 0-5 year duration category was used.[9]

The estimated coefficients of the age of onset/duration dummy variables appear in Table 5. These coefficients suggest that controlling for duration alters the effect of age of onset only in the case of emphysema. For emphysema, when duration is held constant at 0-5 years, age of onset has no effect on participation. Having the disease for 6-10 years, however, significantly reduces the probability of participation. In the case of arteriosclerosis, heart attack and stroke, however, the main effect on labor force participation is caused by age of onset, with onset between 45 and 54 making participation less likely, and onset between 55 and 65 having no significant effect. These results suggest that the effect of age of onset and duration are, in general, disease-specific.

Earnings. The results for our earnings equations suggest that, for the respiratory and coronary diseases studied here, most labor market effects occur through reductions in participation rather than reductions in earnings. Table 6 presents coefficients of the disease dummies in an earnings equation in which diseases are distinguished by age of onset and, in the case of emphysema, by duration.[10] The only respiratory and circulatory diseases studied that significantly reduce earnings are emphysema and heart attack. Having emphysema for 6-10 years reduces earnings by 65%. Having a heart attack between the ages of 45 and 54 reduces earnings by 45%.

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[8] One could, of course, argue that persons with very severe cases of the disease die soon after diagnosis; hence duration may not measure severity.

[9] Persons with an age of onset between 55 and 65 with duration greater than 5 years thus had a value of zero for all health dummies, as did persons without the disease.

The Magnitude of Expected Earnings Losses. The expected loss in earnings to a person who contracts a chronic disease is the sum of the effects of the disease on probability of participation, and on earnings, given that one participates. Specifically, the expected loss in earnings is the sum of the change in probability of participation times pre-illness earnings, plus the reduction in earnings caused by the disease times the post-illness participation rate, $P_1$,

$$\text{Expected Loss in Earnings} = \Delta P(\text{Earnings}_0) + P_1(\Delta\text{Earnings}). \qquad (4)$$

This loss begins at age of onset and continues until the age that retirement would occur in the absence of the disease.

Tables 7 and 8 present estimates of the first term in (4), expected earnings losses due to non-participation. The effect of each disease on probability of participation, $\Delta P$, is determined by multiplying the coefficient of the disease in the participation equation, $\delta_i$, by $P(1-P)$, where $P$ is the probability of being in the labor force. Table 7 presents estimates of $\Delta P$, the fraction by which pre-illness earnings are reduced due to non-participation. In the table, $P$ is estimated at each age from Bureau of Labor Statistics data on labor force participation rates (U.S. Department of Labor, Bureau of Labor Statistics, 1988). In Table 8 $\Delta P$ has been multiplied by average 1987 earnings of all male workers to produce annual earnings losses, by age, due to non-participation.

In both tables earnings losses due to increased probability of not working peak between 55 and 65, because $P(1-P)$ is maximized in this interval. The maximum annual expected reduction in earnings ranges from 15.5% for heart attacks to 57.1% for strokes. Bronchitis and emphysema each reduce expected earnings (through effects on participation) by at most 25%.

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[10] Because fewer chronically ill people appear in the earnings equation than in the participation equation it was necessary to eliminate certain age of onset categories from the earnings equations.

For emphysema, arteriosclerosis, stroke and other heart disease earnings losses due to reduced probability of participation constitute the total change in expected earnings. For emphysema and heart attack the second term in equation (4) must be computed. This term, in $1977, appears in Table 8 together with expected earnings losses due to non-participation.

Comparison with Previous Work. The only study of the labor market effects of chronic respiratory and circulatory diseases of which we are aware is Bartel and Taubman (1979). Using data from the NAS Twins Panel, Bartel and Taubman examine the effects of each of several disease groups on labor force participation and on earnings, conditional on participation. Unfortunately the diseases groupings used by Bartel and Taubman do not correspond exactly to the diseases used in our study. They combine bronchitis, emphysema and asthma into a single disease category (BRON), and heart disease and hypertension into another category (HH). The effect of each disease category is examined for various ages of onset; however, emphasis is placed on diagnoses that occurred between 1962-67, when respondents were in their early forties. Because emphysema, arteriosclerosis and stroke are rare at this age, it is unlikely that BRON and HH capture these more severe diseases.

When they examine the effects of a diagnosis at age 40 on participation at age 50 Bartel and Taubman do not find any significant effects of respiratory or circulatory diseases on labor force participation. This is in sharp contrast to the results presented in Table 7, which indicate that chronic bronchitis, emphysema, arteriosclerosis, heart attack, stroke, and other heart disease reduce the probability of labor force participation between 6 and 57 percentage points. The difference in findings may be due in part to the relatively young age of their sample. The disease variable used in the participation equation represents the effects on participation at (mean) age 50 of a diagnosis that occurred at (mean) age 40. For the diseases we study the most significant effects on participation correspond to an average age of onset of 50.

Regarding effects on earnings, Bartel and Taubman find that a diagnosis of respiratory illness (BRON) at age 40 reduces earnings by 25% at age 50 and that heart disease/hypertension (HH), diagnosed at age 40, reduces earnings by 8.5% at age 50. By contrast, we find that having emphysema for at least 6 years reduces earnings by an average of 65% for persons who continue working. The corresponding reduction in earnings due to having a heart attack between 45 and 54 is 45%. We thus find greater effects on earnings than do Bartel and Taubman, but for more narrowly defined diseases. The difference between our results and theirs reflects the fact that their disease categories include less severe diseases, such as bronchitis and hypertension, as well as more debilitating ones.

## MEDICAL EXPENDITURES AND SERVICES UTILIZATION

The medical costs of a chronic disease to society are the costs of the detection, treatment, and rehabilitation of the disease, as well as a portion of research, training, and facilities costs. In this section we present measures of medical expenditures for individuals for five target diseases: hypertension, ischemic heart disease, non-specific heart disease, chronic bronchitis, and emphysema. These measures were computed from self-provided cost of treatment data for persons in the 1977-78 National Medical Care Expenditure Survey (National Center for Health Services Research, 1981).

There are three reasons why our measures of medical expenditures do not measure the true social costs of medical treatment. First, medical expenditures are computed using market prices, which may not reflect marginal productivities due to the absence of competition in the market for medical services. Second, because the data are specific to individuals with chronic diseases, the costs of detection are not included. In addition, because medical care providers are a minor source of research and medical training, these cost components are likely to be greatly underestimated (if included in overhead charges) or ignored completely.

## The National Medical Care Expenditure Survey

To estimate the medical costs of chronic respiratory and heart disease we used the 1977-78 National Medical Care Expenditure Survey (NMCES). NMCES presents data on health care utilization and expenditures for approximately a one year period for 14,000 households (40,320 persons) selected randomly from the civilian noninstitutionalized U.S. population. Each of these households was provided with a calendar diary for recording their use and cost of medical services. Each was interviewed six times over this period, with responses in prior periods provided to the household for verification.

Each time a person in the NMCES suffered an activity limitation, disability day, visited or called a doctor, went to the hospital or purchased medication a record was created for an illness episode. Information on the number and cost of illness episodes and on the cause of each illness episode comes from the household survey. Medical costs are thus self-reported costs.[11] The diseases associated with each illness episode were reported by households, and translated into ICDA codes by interviewers.

The five respiratory and circulatory diseases we examine, their ICDA codes, and the number of persons reporting episodes involving each condition appear in Table 9.

## Allocation of Medical Costs Among Multiple Conditions

To calculate the costs associated with a target condition one must add the costs associated with the condition across all illness episodes. This would pose no problem if all episodes of illness were associated with only a single disease. If, however, an illness episode is associated with more than one condition, the cost of the episode must be allocated among conditions.

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[11] To check on the accuracy of these costs, the household survey was supplemented by a survey of physicians and facilities that provided medical care to persons in the household sample period and by a survey of employers and insurance companies responsible for the health insurance coverage of responding households. A close correspondence was found between reported and actual costs.

Table 10 indicates the extent of the joint cost allocation problem. The table indicates that of the 3,479 persons with at least one episode of hypertension, 71% (2,476) had episodes that involved hypertension alone. [In the language of NMCES an episode involving only a single condition is a "simple" episode.] For these persons the problem of cost attribution does not arise. Thirteen percent of persons (426 persons) with hypertension episodes have "related to" episodes--episodes that involve hypertension and some other condition. In these cases the respondent attempted to allocate costs among the related conditions; however, in cases where no attribution was possible, for example, the case of hospital room charges, the costs were duplicated for each condition. "Same as" episodes, involving 7% of all persons with hypertension, mean that the individual attributed the episode to hypertension and a condition that was the "same as" hypertension--although it was assigned a different ICDA code. In this case no allocation of costs among the multiple conditions is possible; instead, the total costs of the episode are associated with each condition. "Same as" episodes thus lead to double counting of medical costs, and "related to" episodes may involve some double counting.

The number of persons with "multiple episodes" are found by subtracting those with single episodes from the total (e.g., for hypertension, 314 persons had multiple episodes). In general, persons with more than one episode involving the same disease have other than "simple" episodes that may involve double-counting problems.

## Results

Magnitude of Expenses, by Disease. Table 11 shows the frequency distribution of annual medical expenses for each of our target diseases, as well as mean and median expenses. [All figures are in 1977 dollars.] As one would expect, the highest average expenditures are associated with ischemic heart disease ($1256) and non-specific heart disease ($1041). Emphysema is associated with a mean expenditures of $633. The average annual costs of hypertension and bronchitis are considerably less: $216 and $97, respectively.

In each case the distribution of annual expenses is highly skewed: median expenses are one-quarter of mean expenses for bronchitis and hypertension and approximately one-tenth of mean expenditures for emphysema, ischemic heart disease and non-specific heart disease. For all diseases but ischemic heart disease at least half of all persons have annual expenditures of $75 or less. [For ischemic heart disease 41% of all persons have annual expenditures of $75 or less.]

Categories of Expenses. Table 12 shows how expenditures are distributed across categories for each disease. NMCES allocates expenses to three major categories: medical contacts (primarily doctor visits), hospital expenses, and drugs. There are several minor categories that are omitted from the table.

As would be expected, hospital expenses are the largest category of expenses for all conditions, even when people with no hospital expenses are included in the averaging computation. The maximum hospital expenses per person exceed $20,000 for the heart diseases and are in the $10,000 range for the other target diseases. Expenses on medical contacts are the next largest category of expenses for all conditions.

Comparison With Other Studies. The NHLBI (1982) estimates annual expenditures on chronic bronchitis and emphysema using the "top-down" approach described above while Freeman et al. (1976) use an engineering approach with aggregate data to estimate annual expenditures on emphysema. Table 13 provides the NHLBI and Freeman estimates of total and per person expenditures adjusted to 1977 dollars using the medical price index.

The NHLBI estimates of expenses per case, at $118 and $102 for chronic bronchitis and emphysema, respectively, contrast sharply with ours, at $97 and $633. Nevertheless, because of the top-down nature of the NHLBI approach, their estimates may differ from ours if different estimates of disease prevalence are being used. In fact, the NHLBI prevalence estimates for these diseases (which are taken from the Health Interview Survey (HIS)) are 3.5 and 1.0 percent of the civilian, noninstitutionalized population of the U.S. in 1979 (216 million people) for chronic bronchitis and emphysema, respectively. Our estimates of prevalence, which are conditional on the occurrence of some medical event (i.e., a restricted activity day, some cost incurred, or some service used (including a phone call to the doctor)), are far lower -- 1.1 and 0.5 percent for chronic bronchitis and emphysema, respectively, for 1977.

The underestimate of prevalence implied by this conditionality implies that our sample would under-represent, relative to the NHLBI, people with zero medical costs. This implies, in turn, that the NHLBI estimate of expense per case should be lower than ours. Instead, the NHLBI estimate for chronic bronchitis, the disease for which the highest proportion of sufferers in our sample has zero costs, actually **exceeds** our estimate.

Freeman et al, using data on health care utilization and average prices for 1970, estimate expenses on emphysema in 1977 dollars of $233.5 per case annually. These estimates are over double those of the NHLBI but still are far lower than ours.

Sources of Payment. NMCES provides information on five sources of funding for medical expenses: family, medicaid, medicare, personal insurance, and other. In addition to being of intrinsic interest, information about sources of funding suggests the extent to which medical costs are likely to be internalized in willingnesses to pay to avoid disease. In theory, willingness to pay should take into account the medical costs of the condition paid for by the family, but not those costs borne by others. Thus, the portion of expenses paid by others should be added to the bid as part of the social cost of each of the target conditions.

Table 14 identifies these funding sources by condition for males 20 years of age and older, the group to which our labor market analysis applies. For each disease the second row of the table gives the percent of total costs paid for by each source. Even for hypertension and bronchitis, the least serious diseases studied, families pay a minority of total costs, 23% and 34%, respectively. For emphysema and the heart diseases families pay less than 15% of total costs. What are the most important sources of funding? Personal insurance is the most important source of funding for ischemic heart disease (46 percent), reflecting the high proportion of expenses for the hospitalization component and the high degree of coverage afforded this type of expense by health insurance plans. The insurance share for emphysema is large (28 percent) for much the same reason. Coverage for non-specific heart disease, the condition with the least family funding, is not dominated by insurance. Rather, because the population with this condition tends to be older than that for ischemic heart disease, the largest funding share comes from medicare (36 percent). Finally, it is curious that medicaid funds less than one percent of expenses for ischemic heart disease while funding from 7 to 17 percent of the expenses for the other target conditions.

Although a minority of total medical costs are paid for directly by patients and their families, family funding is the most important source of payment for a majority of patients. This is because most patients incur small expenses (see Table 11) and families bear a larger percent of small expenses than of large expenses. For each disease the third row of Table 14 computes for each individual the percentages of funding received from various sources and then averages these percentages across individuals for each source. As can be seen, the average percentages for the family source (in brackets) are much higher than the aggregate percentages for the family source (in parentheses), the former ranging from 52 to 70 percent, while the latter ranges from 13 to 36 percent. This difference implies that relatively large numbers of people have episodes with small expenses that they pay for themselves. This may reflect deductibility clauses, the exclusion of drugs from coverage for some policies, or other factors.

Age Distribution of Expenses. To permit comparison of the labor market effects of chronic respiratory and circulatory diseases with medical costs, Table 15 presents average medical costs for males, by age. Mean annual expenses appear generally to increase with age, up to the '60's or

70's for bronchitis, emphysema and hypertension. Expenses for those with heart disease (heart attacks), however, peak in the '40's.

A comparison of average medical expenses with the labor market effects of each chronic disease (see Table 8) suggests that the labor market costs of chronic respiratory and circulatory diseases are generally greater than the medical costs. Exceptions to this result are hypertension, which has no effect on labor force participation or on earnings, and heart disease before the age of 45, which also appears to have no significant labor market effects.

Table 1. Non-health Variables Entering Earnings and Participation Equations

| | Mean | Standard deviation | Maximum | Minimum |
|---|---|---|---|---|
| Earnings, 1977* | 14,362. | 77045. | 50000. | 0 |
| In labor force, 1977 | 0.670 | | 1 | 0 |
| Married[a] | 0.718 | 0.45 | 1 | 0 |
| No. in household[a] | 3.294 | 1.732 | 15 | 1 |
| No. children < 5[a] | 0.190 | 0.512 | 5 | 0 |
| No. children 5-18[a] | 0.670 | 1.174 | 8 | 0 |
| No. children > 18[a] | 0.184 | 0.482 | 3 | 0 |
| Age dummies: | | | | |
| 18-24 | 0.141 | 0.348 | 1 | 0 |
| 35-44 | 0.174 | 0.379 | 1 | 0 |
| 45-54 | 0.222 | 0.416 | 1 | 0 |
| 55-65 | 0.261 | 0.440 | 1 | 0 |
| Highest educ. level: | | | | |
| Elementary school | 0.193 | 0.394 | 1 | 0 |
| High school | 0.487 | 0.500 | 1 | 0 |
| College | 0.229 | 0.421 | 1 | 0 |
| Non-white | 0.124 | 0.330 | 1 | 0 |
| Regional dummies[a]: | | | | |
| Northcentral | 0.265 | 0.441 | 1 | 0 |
| South | 0.335 | 0.472 | 1 | 0 |
| West | 0.178 | 0.383 | 1 | 0 |
| Lives in Urban Area[a] | 0.679 | 0.467 | 1 | 0 |
| $(\text{Age}-16)^2$ | 888.25 | 730.23 | 2401 | 4 |
| Veteran | 0.452 | 0.498 | 1 | 0 |
| Aware of disability benefits | 0.407 | 0.491 | 1 | 0 |
| Debt[a] | 2116.9 | 8858.00 | 200800 | 0 |

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*Average based on 1486 persons in labor force

[a] Measured as of interview date

Table 2. Health Variables in Earnings and Participation Equations

Each of the following variables assume a value of 1 if the respondent contracted the disease at the age indicated and a value of 0 otherwise:

**RESPIRATORY AND CIRCULATORY DISEASES**

| | **Age of Onset Categories (Sample Size)** | | | |
|---|---|---|---|---|
| Allergies | 0-17 (35) | 18-34 (37) | 35-65 (18) | |
| Asthma | 0-17 (40) | 18-34 (14) | 35-65 (19) | |
| Chronic Bronchitis | 25-44 (18) | 45-65 (21) | | |
| Emphysema | 45-54 (49) | 55-65 (23) | | |
| Other Chronic Lung Dis. | 18-44 (17) | 45-65 (26) | | |
| Arteriosclerosis | 45-54 (55) | 55-65 (24) | | |
| Heart Attack | 25-44 (28) | 45-54 (57) | 55-65 (42) | |
| Hypertension | 25-34 (57) | 35-44 (79) | 45-54 (148) | 55-65 (66) |
| Other Chronic Heart Disease | 0-34 (23) | 35-44 (34) | 45-54 (51) | 55-65 (22) |
| Stroke | 45-54 (17) | 55-65 (20) | | |

| **OTHER CHRONIC DISEASES** | **Sample Size** |
|---|---|
| Arthritis or rheumatism | 367 |
| Other trouble with back or spine | 296 |
| Deformity of foot, leg, arm, hand | 228 |
| Nervous or emotional problems | 209 |
| Deformity of back or spine | 154 |
| Deafness | 133 |
| Stomach ulcer | 130 |
| Diabetes | 113 |
| Hernia or rupture | 92 |
| Difficulty reading (with glasses) | 86 |
| Kidney stones or kidney trouble | 76 |
| Other chronic stomach trouble | 64 |
| Tumor, cyst or growth | 52 |
| Missing arms, hands or fingers | 46 |
| Gallbladder or liver trouble | 40 |
| Paralysis | 35 |
| Alcohol or drug problems | 25 |
| Cancer | 24 |
| Epileptic seizures | 24 |
| Mental illness | 20 |
| Blindness | 19 |
| Thyroid trouble or goiter | 18 |
| Missing legs or feet | 14 |
| Tuberculosis | 7 |
| Multiple sclerosis | 6 |

Table 3. Distribution of Respiratory and Circulatory Diseases by Age of Onset

| | Number of persons in sample with age of onset | | | | | |
|---|---|---|---|---|---|---|
| | 0-17 | 18-24 | 25-34 | 35-44 | 45-54 | 55-65 |
| Allergies | 35 | 18 | 19 | 10 | 4 | 4 |
| Asthma | 40 | 5 | 9 | 7 | 9 | 3 |
| Chronic Bronchitis | 15 | 2 | 13 | 5 | 15 | 6 |
| Emphysema | 0 | 1 | 4 | 3 | 49 | 23 |
| Other Chronic Lung Diseases | 1 | 4 | 7 | 6 | 20 | 6 |
| Arteriosclerosis | 0 | 0 | 7 | 11 | 55 | 24 |
| Heart Attack | 2 | 0 | 5 | 23 | 57 | 42 |
| Hypertension | 12 | 23 | 57 | 79 | 148 | 66 |
| Other Chronic Heart Disease | 18 | 5 | 10 | 34 | 51 | 22 |
| Stroke | 1 | 0 | 2 | 2 | 17 | 20 |

Table 4. Effects of Chronic Diseases on Labor Force Participation by Age of Onset

| | Age of onset | Coefficient | \|t-Ratio\| |
|---|---|---|---|
| Asthma | 0-17 | 0.093 | 0.22 |
| | 18-34 | 0.625 | 0.75 |
| | 35-65 | 0.093 | 0.16 |
| Allergies | 0-17 | -0.061 | 0.13 |
| | 18-34 | 0.505 | 0.95 |
| | 35-65 | -0.565 | 0.91 |
| Chronic Bronchitis | 25-44 | -1.229 | 1.69 |
| | 45-65 | -0.816 | 1.17 |
| Emphysema | 45-54 | -1.053 | 2.55 |
| | 55-65 | -0.683 | 1.21 |
| Other Chronic Lung Disease | 18-44 | -0.218 | 0.29 |
| | 45-65 | -0.528 | 0.95 |
| Arteriosclerosis | 45-54 | -0.707 | 1.72 |
| | 55-65 | 0.134 | 0.26 |
| Hypertension | 25-34 | -0.435 | 1.16 |
| | 35-44 | -0.131 | 0.38 |
| | 45-54 | 0.189 | 0.78 |
| | 55-65 | -0.112 | 0.34 |
| Heart Attack | 25-44 | -0.463 | 0.94 |
| | 45-54 | -0.720 | 1.94 |
| | 55-65 | 0.507 | 1.15 |
| Stroke | 45-54 | -2.593 | 2.38 |
| | 55-65 | -1.530 | 1.41 |
| Other Heart Disease | 0-34 | -0.393 | 0.90 |
| | 35-44 | -0.184 | 0.40 |
| | 45-54 | -0.896 | 2.39 |
| | 55-65 | -1.462 | 2.04 |

Table 5. Effects of Chronic Diseases on Labor Force Participation by Duration of Disease and Age of Onset

| | Duration | Onset | Coefficient | \|t-Ratio\| |
|---|---|---|---|---|
| Asthma | | | | |
| | | 0-17 | 0.017 | 0.04 |
| | | 18-34 | 0.780 | 0.92 |
| | | 35-65 | 0.029 | 0.05 |
| Allergies | | | | |
| | | 0-17 | -0.040 | 0.09 |
| | | 18-34 | 0.542 | 1.02 |
| | | 35-65 | -0.479 | 0.78 |
| Chronic Bronchitis | | | | |
| | | 25-44 | -1.254 | 1.70 |
| | | 45-65 | -1.013 | 1.46 |
| Emphysema | | | | |
| | 0-5 | 45-54 | -0.230 | 0.35 |
| | 5-10 | 45-54 | -1.299 | 2.04 |
| | 0-5 | 55-65 | -0.370 | 0.62 |
| Other Chronic Lung Diseases | | | | |
| | | 18-44 | -0.465 | 0.65 |
| | | 45-65 | -0.670 | 1.19 |
| Arteriosclerosis | | | | |
| | 0-5 | 45-54 | -0.389 | 0.57 |
| | 5-10 | 45-54 | -0.252 | 0.41 |
| | 0-5 | 55-65 | 0.659 | 1.11 |
| Hypertension | | | | |
| | | 25-34 | -0.418 | 1.12 |
| | | 35-44 | -0.151 | 0.44 |
| | | 45-54 | 0.084 | 0.35 |
| | | 55-65 | -0.088 | 0.27 |
| Heart Attack | | | | |
| | | 25-44 | -0.449 | 0.91 |
| | 0-5 | 45-54 | -1.003 | 1.70 |
| | 5-10 | 45-54 | -1.069 | 1.85 |
| | 0-5 | 55-65 | 0.371 | 0.79 |
| Stroke | | | | |
| | 0-5 | 45-54 | -1.503 | 1.25 |
| | 5-10 | 45-54 | -7.551 | 0.38 |
| | 0-5 | 55-65 | -0.900 | 1.06 |
| Other Heart Disease | | | | |
| | | 0-34 | -0.352 | 0.81 |
| | | 35-44 | -0.165 | 0.36 |
| | 0-5 | 45-54 | -1.119 | 1.75 |
| | 5-10 | 45-54 | -0.007 | 0.01 |
| | 0-5 | 55-65 | -1.273 | 1.73 |

Table 6. Effects of Chronic Diseases on Ln(Earnings) by Age of Onset

| | Age of Onset | Coefficient | \|T-Ratio\| |
|---|---|---|---|
| Asthma | - | -0.232 | 1.020 |
| Allergies | - | -0.061 | 0.318 |
| Chronic Bronchitis | - | -0.023 | 0.065 |
| Emphysema | 0-5[a] | 0.229 | 0.641 |
| | 6-10[a] | -1.038 | 2.009 |
| Other Chronic Lung Disease | - | -0.511 | 1.294 |
| Arteriosclerosis | 45-54 | 0.279 | 0.680 |
| | 55-65 | -0.624 | 1.510 |
| Hypertension | 25-34 | 0.207 | 0.916 |
| | 35-44 | -0.041 | 0.188 |
| | 45-54 | 0.193 | 1.211 |
| | 55-65 | 0.311 | 1.167 |
| Heart Attack | 25-44 | 0.056 | 0.151 |
| | 45-54 | -0.590 | 1.706 |
| | 55-65 | -0.376 | 1.141 |
| Stroke | - | 0.843 | 1.386 |
| Other Heart Disease | 35-44 | 0.302 | 1.008 |
| | 45-54 | 0.055 | 0.165 |

[a] Denotes duration of disease rather than age of onset.

Table 7. Effect of Respiratory and Circulatory Diseases on Probability of Participation by Age of Onset

| Disease | Age of Onset | Change in probability of participation at each age | | | | |
|---|---|---|---|---|---|---|
| | | 25-34 | 35-44 | 45-54 | 55-65 | 65+ |
| Chronic Bronchitis | 25 | -0.067 | -0.067 | -0.111 | -0.288 | -0.180 |
| | 45 | | | -0.084 | -0.218 | -0.136 |
| Emphysema | 45 | | | -0.099 | -0.256 | -0.159 |
| Arteriosclerosis | 45 | | | -0.060 | -0.157 | -0.098 |
| Heart Attack | 45 | | | -0.059 | -0.155 | -0.096 |
| Stroke | 45 | | | -0.220 | -0.571 | -0.356 |
| | 55 | | | | -0.327 | -0.204 |
| Other Heart Disease | 45 | | | -0.075 | -0.196 | -0.122 |
| | 55 | | | | -0.324 | -0.202 |

Table 8. Annual Change in Expected Earnings at Each Age Due to Various Chronic Diseases ($1977)

| | | Annual Change Due to Reduced Probability of Participation (Change Due to Reduction in Earnings if Working) | | | | |
|---|---|---|---|---|---|---|
| Disease | Age of onset | 25-34 | 35-44 | 45-54 | 55-65 | 65+ |
| Chronic Bronchitis | 25 | $-870.2 | $-1226.3 | $-2229.1 | $-4860.9 | $-1680.4 |
| | 45 | | | -1689.6 | -3684.4 | -1273.7 |
| Emphysema[a] | 45 | | | -1978.4 | -4314.3<br>(-10891.) | -1491.5<br>(-6044.7) |
| Arteriosclerosis | 45 | | | -1210.6 | -2639.9 | -912.6 |
| Heart Attack | 45 | | | -1197.7<br>(-8949.6) | -2611.8<br>(-7515.8) | -902.9<br>(-4171.2) |
| Stroke | 45 | | | -4415.8 | -9629.5 | -3328.9 |
| | 55 | | | | -5511.0 | -1905.2 |
| Other Heart Disease | 45 | | | -1513.7 | -3301.0 | -1141.2 |
| | 55 | | | | -5455.6 | -1886.0 |

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[a] Effects on Earnings do not begin until duration is greater than or equal to 6 years.

Table 9. Sample size by condition, NMCES.

| Disease | ICDA codes | Persons |
|---|---|---|
| Total | | 4789 |
| Hypertension | 401-404 | 3479 |
| Ischemic heart disease | 410-414 | 378 |
| Non-specific heart disease | 429 | 884 |
| Chronic bronchitis | 490-491 | 430 |
| Emphysema | 492 | 222 |

Table 10. Distribution of single vs multiple episodes types.

| Disease | Total persons | Number of persons with single episodes: One simple | One same-as | One* related-to/ stand-alone | Percent with only one single episode |
|---|---|---|---|---|---|
| Hypertension | 3479 | 2476 | 227 | 462 | 91.0 |
| Ischemic | 378 | 195 | 34 | 80 | 81.7 |
| Non-specific heart | 884 | 501 | 104 | 166 | 87.2 |
| Chronic bronchitis | 430 | 272 | 49 | 63 | 89.3 |
| Emphysema | 222 | 130 | 21 | 42 | 86.9 |

*In each of these cases there is only one 'stand alone' episode to analyze that is associated with our target disease.

Table 11. Frequency Distribution of Annual Expenses per Person, by Condition. Unweighted.

| Total Expense ($1977) | Percentage of Sample in Each Expense Category | | | | |
|---|---|---|---|---|---|
| | Bronchitis | Emphysema | Hypertension | Ischemic HD | Nonspecific HD |
| $ 0 | 17.4 | 20.7 | 6.7 | 9.0 | 12.9 |
| 0-25 | 36.3 | 23.0 | 21.8 | 15.9 | 19.2 |
| 25-50 | 19.8 | 7.7 | 19.2 | 10.3 | 11.5 |
| 50-75 | 8.4 | 5.4 | 13.2 | 5.6 | 6.8 |
| 75-100 | 4.7 | 4.5 | 8.7 | 6.3 | 5.1 |
| 100-150 | 5.1 | 8.6 | 11.2 | 9.5 | 7.0 |
| 150-200 | 1.6 | 4.1 | 5.5 | 6.9 | 4.6 |
| 200-300 | 3.0 | 5.4 | 5.1 | 9.3 | 7.6 |
| 300-400 | 0.7 | 1.8 | 2.4 | 2.6 | 3.2 |
| 400-500 | 0.5 | 0.9 | 1.2 | 2.4 | 1.6 |
| 500-750 | 0.9 | 2.7 | 1.3 | 2.4 | 2.5 |
| 750-1000 | 0.2 | 1.4 | 0.6 | 2.6 | 1.5 |
| 1000-1500 | 0.5 | 3.6 | 1.1 | 2.9 | 2.6 |
| 1500-2000 | 0.2 | 4.1 | 0.4 | 2.4 | 1.9 |
| 2000-3000 | 0.2 | 1.4 | 0.6 | 1.3 | 2.6 |
| 3000-4000 | . | 0.9 | 0.2 | 2.9 | 3.1 |
| 4000-5000 | 0.2 | 1.4 | 0.1 | 0.3 | 1.4 |
| 5000-10000 | 0.2 | 1.4 | 0.5 | 3.4 | 2.9 |
| 10000-20000 | . | 1.4 | 0.1 | 2.6 | 1.1 |
| 20000+ | . | . | 0.1 | 1.3 | 0.9 |
| N | 430 | 222 | 3479 | 378 | 884 |
| Mean Expense | $96.74 | $632.76 | $215.79 | $1257.55 | $1041.26 |
| Median Expense | $23.27 | $42.63 | $53.51 | $116.26 | $73.90 |

Table 12. Average Expenses Per Person By Disease and Category ($1977).

| CONDITION | Expense | Mean Expense | Std Dev | Maximum |
|---|---|---|---|---|
| Bronchitis (n=430) | Medical Contact | $38.87 | $117.30 | $1683.00 |
| | Hospital | 41.30 | 499.76 | 9635.00 |
| | Drugs | 14.65 | 44.60 | 605.27 |
| | Total Expense | 96.74 | 537.54 | 9712.00 |
| Emphysema (n=222) | Medical Contact | 72.06 | 179.13 | 1683.00 |
| | Hospital | 498.40 | 2073.30 | 18832.00 |
| | Drugs | 46.43 | 94.72 | 730.01 |
| | Total Expense | 632.76 | 2171.28 | 19563.78 |
| Hypertension (n=3479) | Medical Contact | 51.88 | 127.12 | 2854.89 |
| | Hospital | 111.65 | 1278.68 | 57940.00 |
| | Drugs | 41.62 | 55.89 | 970.45 |
| | Total Expense | 215.79 | 1377.29 | 60588.00 |
| Ischemic HD (n=378) | Medical Contact | 96.23 | 273.83 | 3977.33 |
| | Hospital | 1069.38 | 3653.32 | 35910.00 |
| | Drugs | 68.88 | 105.86 | 791.32 |
| | Total Expense | 1257.55 | 3831.66 | 36462.00 |
| Nonspecific HD (n=884) | Medical Contact | 82.45 | 220.92 | 4074.04 |
| | Hospital | 859.10 | 3479.98 | 49638.00 |
| | Drugs | 44.71 | 83.18 | 1094.67 |
| | Total Expense | 1041.26 | 3736.60 | 49743.00 |

Table 13. Medical Expenses on Chronic Bronchitis and Emphysema from the NHLBI (1982) and Freeman et al (1976). (1977 $'s)

NHLBI

| | Hospital | Doctor | Drugs | Total |
|---|---|---|---|---|
| Chronic Bronchitis (millions of $'s) | $285 | $162 | $432 | $879 |
| Per Person | (38.1) | (57.8) | (21.7) | (117.7) |
| Emphysema (millions of $'s) | 152 | 48 | 19 | 219 |
| Per Person | (71.0) | (22.5) | (8.7) | (102.1) |

Freeman et al

| | Hospital | Doctor | Drugs | Total |
|---|---|---|---|---|
| Emphysema (millions of $'s) | $174 | $71 | $59 | $304 |
| Per Person | (133.4) | (54.5) | (45.6) | (233.5) |

Table 14. Funding Source by Condition for Males 20 Years of Age or Greater. Weighted.

| CONDITION | N[a] | Mean Expense[b] | Family | Medicaid | Medicare | Personal Insurance | Other |
|---|---|---|---|---|---|---|---|
| Bronchitis | 478447 | $205.24 | $69.01<br>(34%)[c]<br>[65%][d] | $4.25<br>(2%)<br>[1%] | $57.58<br>(28%)<br>[4%] | $62.38<br>(30%)<br>[25%] | $12.02<br>(6%)<br>[4%] |
| Emphysema | 766736 | 726.78 | 100.54<br>(14%)<br>[51%] | 96.62<br>(13%)<br>[3%] | 172.74<br>(24%)<br>[12%] | 165.93<br>(23%)<br>[18%] | 190.95<br>(26%)<br>[13%] |
| Hypertension | 6644806 | 268.87 | 60.96<br>(23%)<br>[68%] | 14.44<br>(5%)<br>[3%] | 94.03<br>(35%)<br>[4%] | 48.57<br>(18%)<br>[16%] | 50.87<br>(19%)<br>[9%] |
| Ischemic HD | 1184816 | 1739.77 | 180.77<br>(10%)<br>[50%] | 186.23<br>(11%)<br>[4%] | 287.79<br>(17%)<br>[9%] | 840.05<br>(48%)<br>[28%] | 244.93<br>(14%)<br>[8%] |
| Nonspecific HD | 2019627 | 1662.99 | 164.38<br>(10%)<br>[51%] | 72.77<br>(4%)<br>[6%] | 685.51<br>(41%)<br>[12%] | 493.92<br>(30%)<br>[18%] | 246.41<br>(15%)<br>[13%] |

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[a] Complex Multiple Episode excluded (see text).

[b] Mean does not include observations reporting zero.

[c] Percentage of Mean Expense.

[d] Percentage of Expense by Source, Averaged Over All Individuals.

Table 15. Average Medical Expenses for Males, by Age. Weighted.

| CONDITION | Age Group | N | Mean Expense | Std Dev | Maximum | Total Expense (millions $) |
|---|---|---|---|---|---|---|
| Bronchitis | 0-9 | 438016 | $59.59 | $109.84 | $626.45 | $26.1 |
| | 10-19 | 160828 | 33.55 | 51.83 | 270.00 | 5.4 |
| | 20-29 | 89507 | 84.99 | 148.34 | 514.00 | 7.6 |
| | 30-39 | 60767 | 46.86 | 63.46 | 197.56 | 2.8 |
| | 40-49 | 65470 | 96.40 | 140.54 | 446.05 | 6.3 |
| | 50-59 | 67189 | 141.66 | 186.04 | 654.60 | 9.5 |
| | 60-69 | 125470 | 249.56 | 781.04 | 4251.16 | 31.3 |
| | 70-79 | 58254 | 485.20 | 2061.74 | 9712.00 | 28.3 |
| | 80-89 | 11790 | 60.22 | 51.86 | 116.00 | 0.7 |
| | ≥ 20 | 478447 | 180.94 | 841.34 | 9712.00 | 86.6 |
| | Average | 1077291 | 109.60 | 569.05 | 9712.00 | 118.1 |
| Emphysema | 40-49 | 50017 | 562.54 | 672.46 | 1647.00 | 28.1 |
| | 50-59 | 164485 | 884.41 | 2481.30 | 13535.82 | 145.5 |
| | 60-69 | 341324 | 371.82 | 1639.63 | 17615.01 | 126.9 |
| | 70-79 | 168861 | 580.66 | 2667.20 | 19563.78 | 98.1 |
| | 80-89 | 39177 | 1474.26 | 1585.82 | 4854.75 | 57.8 |
| | 90-99 | 2872 | 2.19 | 0.00 | 2.19 | 0.01 |
| | ≥ 20 | 766736 | 595.16 | 2079.27 | 19563.78 | 456.3 |
| | Average | 766736 | 595.16 | 2097.27 | 19563.78 | 456.3 |
| Hypertension | 0-9 | 17632 | 37.16 | 51.85 | 132.14 | 0.7 |
| | 10-19 | 42691 | 241.54 | 436.87 | 1186.45 | 10.3 |
| | 20-29 | 266550 | 74.09 | 199.29 | 1852.00 | 19.7 |
| | 30-39 | 563863 | 96.74 | 369.69 | 6427.20 | 54.6 |
| | 40-49 | 1000099 | 183.71 | 627.80 | 5504.85 | 183.7 |
| | 50-59 | 1720562 | 264.21 | 1502.78 | 22771.07 | 454.6 |
| | 60-69 | 1763206 | 486.97 | 3950.75 | 60588.00 | 858.6 |
| | 70-79 | 1025353 | 115.82 | 428.34 | 9144.00 | 118.8 |
| | 80-89 | 343210 | 176.83 | 358.22 | 2391.58 | 60.7 |
| | 90-99 | 21317 | 80.95 | 63.68 | 140.70 | 1.7 |
| | 100+ | 5215 | 37.80 | 0.00 | 37.80 | 0.2 |
| | ≥ 20 | 6709375 | 261.22 | 2192.97 | 60588.00 | 1752.6 |
| | Average | 6769698 | 260.15 | 2183.48 | 60588.00 | 1763.6 |
| Ischemic HD | 10-19 | 4014 | 0.00 | 0.00 | 0.00 | 0.0 |
| | 30-39 | 21589 | 102.33 | 99.31 | 239.82 | 2.2 |
| | 40-49 | 138574 | 4691.54 | 8048.54 | 23840.63 | 650.1 |
| | 50-59 | 416557 | 1346.61 | 2772.72 | 14697.77 | 560.9 |
| | 60-69 | 381771 | 1556.08 | 4631.68 | 23413.50 | 594.1 |
| | 70-79 | 187042 | 769.57 | 2370.64 | 12571.90 | 143.9 |
| | 80-89 | 74932 | 1174.44 | 3013.67 | 11320.83 | 88.0 |
| | ≥ 20 | 1220465 | 1670.91 | 4400.88 | 23840.63 | 2039.3 |
| | Average | 1224479 | 1665.43 | 4394.70 | 23840.63 | 2039.3 |
| Nonspecific HD | 0-9 | 4451 | 0.00 | 0.00 | 0.00 | 0.0 |
| | 10-19 | 18671 | 402.77 | 763.10 | 2009.00 | 7.5 |
| | 20-29 | 41827 | 974.21 | 1958.24 | 4966.51 | 40.7 |

| | | | | | |
|---|---|---|---|---|---|
| 30–39 | 20574 | 1063.75 | 1614.51 | 3543.00 | 21.9 |
| 40–49 | 204956 | 2032.02 | 4906.42 | 23883.04 | 416.5 |
| 50–59 | 524168 | 1375.39 | 5077.84 | 38375.75 | 720.9 |
| 60–69 | 595206 | 1274.62 | 4634.08 | 43326.75 | 758.7 |
| 70–79 | 426144 | 2224.78 | 7825.51 | 49743.00 | 948.1 |
| 80–89 | 202381 | 589.05 | 2070.29 | 15360.86 | 119.2 |
| 90–99 | 37315 | 276.94 | 450.81 | 1194.00 | 10.3 |
| 100+ | 5215 | 73.30 | 0.00 | 73.30 | 0.3 |
| ≥ 20 | 2057786 | 1475.72 | 5353.93 | 49743.00 | 3036.7 |
| Average | 2080908 | 1462.93 | 5325.98 | 49743.00 | 3044.2 |

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# APPENDIX

Table A.1 Coefficients of Non-Health Variables in Participation Equation

| | Coefficient | \|t-Ratio\| |
|---|---|---|
| Married[a] | 0.8989 | 5.89 |
| No. in household | -0.1290 | 2.58 |
| No. children < 5[a] | 0.4072 | 2.56 |
| No. children 5-18[a] | 0.1060 | 1.34 |
| No. children > 18[a] | 0.3216 | 2.23 |
| Age dummies: | | |
| 18-24 | -1.2822 | 5.92 |
| 35-44 | 1.1440 | 4.28 |
| 45-54 | 1.5330 | 3.75 |
| 55-65 | 2.2198 | 3.55 |
| Highest educ. level: | | |
| Elementary school | -0.2006 | 0.84 |
| High school | 0.1312 | 0.65 |
| College | 0.0386 | 1.38 |
| Nonwhite | -0.5886 | 3.39 |
| Regional dummies[a]: | | |
| Northcentral | 0.3662 | 2.17 |
| South | -0.1020 | 0.64 |
| West | -0.0808 | 0.45 |
| Lives in Urban Area[a] | 0.1852 | 1.46 |
| $(\text{Age}-16)^2$ | -0.00160 | 4.54 |
| Veteran | -0.1077 | 0.81 |
| Aware of disability benefits | -1.0358 | 8.68 |
| Debt[a] | 0.00004 | 2.56 |

[a] Measured as of interview date

Table A.2 Coefficients of Remaining Health Variables in Participation Equation

| Disease | Coefficient | \|t-Ratio\| |
|---|---|---|
| Arthritis or rheumatism | -0.2791 | 1.65 |
| Other trouble with back or spine | -0.4597 | 2.79 |
| Deformity of foot, leg, arm, hand | -0.3741 | 1.89 |
| Nervous or emotional problems | -0.8574 | 4.10 |
| Deformity of back or spine | -0.7925 | 3.53 |
| Deafness | -0.2624 | 1.08 |
| Stomach ulcer | -0.2714 | 1.11 |
| Diabetes | -0.1334 | 0.49 |
| Hernia or rupture | 0.005837 | 0.02 |
| Difficulty reading (with glasses) | -0.2017 | 0.65 |
| Kidney stones or kidney trouble | -0.1528 | 0.48 |
| Other chronic stomach trouble | -0.2896 | 0.85 |
| Tumor, cyst or growth | 0.1030 | 0.27 |
| Missing arms, hands or fingers | -0.5395 | 1.42 |
| Gallbladder or liver trouble | -1.1440 | 2.40 |
| Paralysis | -1.9011 | 3.49 |
| Alcohol or drug problems | -1.4264 | 2.46 |
| Cancer | -0.82301 | 1.56 |
| Epileptic seizures | -1.5235 | 2.18 |
| Mental illness | -1.0498 | 1.60 |
| Blindness | 0.1043 | 0.16 |
| Thyroid trouble or goiter | -0.2380 | 0.39 |
| Missing legs or feet | -0.5794 | 0.84 |
| Tuberculosis | 0.1099 | 0.09 |
| Multiple sclerosis | -2.3758 | 1.78 |

Table A.3 Coefficients of Non-Health Variables in Earnings Equation

| | Coefficient | \|T-Ratio\| |
|---|---|---|
| Married$^a$ | 0.267 | 2.439 |
| No. in household | - 0.071 | 1.736 |
| No. children < 5$^a$ | 0.050 | 0.634 |
| No. children 5-18$^a$ | 0.058 | 1.117 |
| No. children > 18$^a$ | 0.003 | 0.034 |
| Age dummies: | | |
| 18-24 | - 0.421 | 2.771 |
| 35-44 | 0.230 | 1.601 |
| 45-54 | 0.229 | 0.936 |
| 55-65 | 0.364 | 0.941 |
| Highest educ. level: | | |
| Elementary school | - 0.096 | 0.644 |
| High school | 0.004 | 0.036 |
| College | 0.294 | 2.271 |
| Nonwhite | - 0.195 | 1.550 |
| Regional dummies$^a$: | | |
| Northcentral | 0.111 | 1.136 |
| South | 0.011 | 0.113 |
| West | - 0.025 | 0.231 |
| Lives in Urban Area$^a$ | 0.117 | 1.527 |
| $(Age-16)^2$ | - 0.0002 | 0.806 |

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[a] Measured as of interview date

Note: Dependent variable is ln(earnings).

Table A.4 Coefficients of Remaining Health Variables in Earnings Equation

| Disease | Coefficient | \|t-Ratio\| |
|---|---|---|
| Arthritis or rheumatism | -0.051 | 0.415 |
| Other trouble with back or spine | -0.033 | 0.296 |
| Deformity of foot, leg, arm, hand | 0.043 | 0.301 |
| Nervous or emotional problems | -0.208 | 1.075 |
| Deformity of back or spine | -0.297 | 1.597 |
| Deafness | -0.226 | 1.257 |
| Stomach ulcer | -0.031 | 0.174 |
| Diabetes | -0.300 | 1.690 |
| Hernia or rupture | 0.059 | 0.290 |
| Difficulty reading (with glasses) | -0.136 | 0.520 |
| Kidney stones or kidney trouble | -0.341 | 1.409 |
| Other chronic stomach trouble | 0.242 | 0.954 |
| Tumor, cyst or growth | -0.327 | 1.368 |
| Missing arms, hands or fingers | 0.354 | 1.340 |
| Gallbladder or liver trouble | 0.290 | 0.604 |
| Paralysis | -2.931 | 4.518 |
| Alcohol or drug problems | 0.355 | 0.594 |
| Cancer | -1.003 | 2.215 |
| Epileptic seizures | -1.865 | 2.795 |
| Mental illness | -0.010 | 0.015 |
| Blindness | -0.001 | 0.002 |
| Thyroid trouble or goiter | -0.044 | 0.100 |
| Missing legs or feet | 0.356 | 0.580 |
| Tuberculosis | 0.184 | 0.246 |
| Multiple sclerosis | 0.653 | 0.506 |

# ESTIMATING THE VALUE OF AVOIDING MORBIDITY AND MORTALITY FROM FOODBORNE ILLNESSES

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# ESTIMATING THE VALUE OF AVOIDING MORBIDITY AND MORTALITY FROM FOODBORNE ILLNESSES

## I INTRODUCTION

All foods produced for human consumption in the United States are regulated for composition, quality, safety, and labeling under the Food, Drug, and Cosmetic (FD&C) Act of 1938 and its subsequent amendments. One of the chief goals of the FD&C Act is to reduce the presence of contaminants or adulterants in domestic and imported foods. Consuming foods that contain illegal contaminants or adulterants increases the risk of foodborne illness and decreases consumer welfare. The Food and Drug Administration (FDA) is empowered to ensure compliance of the FD&C Act for all domestic and imported food products. FDA's compliance monitoring program and enforcement activities reduce the probability of violative products reaching consumers and causing welfare losses.

FDA's objective is to maximize social welfare subject to a given compliance monitoring budget. The optimal solution is to allocate program resources across different inspection and enforcement activities to the point where the incremental value per unit expenditures for all activities are equal. To develop such an efficient compliance monitoring program, FDA must consider the costs and benefits of different alternatives. The costs of such programs consist primarily of the value of resources used to inspect and test products, and ensure compliance. The benefits of compliance monitoring activities depend on:

- the impact of compliance acitivities on the probability that violative products will reach the consumer,
- the probability that each violation will lead to various adverse health effects (e.g. salmonellosis, botulism, cancer, or chemical poisoning), and
- the value of the welfare losses associated with each adverse effect.

Figure 1 shows how estimates of the three factors noted above can be combined to estimate the benefits of different compliance monitoring options. Calculating these values is not a straightforward task, however, and requires careful analysis. For example, the impact of compliance activities on the probability of a violative product reaching the consumer depends both on the initial probability of the product violating the FD&C Act as well as on how effectively the compliance monitoring and enforcement activity reduce this probability. The probability of a product violating the Act may vary over time and with country of origin.

The probability that any violation of the Act adversely affects a consumer will depend on the type and degree of the violation. Food contaminated with salmonella will be more likely to have an adverse effect on a consumer if the level of contamination is high, if the typical portion size is large, and if the product is eaten without further cooking. Furthermore, the relationship between dose and the probability of an adverse response may vary for different violations of the Act. For example, the probability of an adverse health effect associated with frequently ingested levels of salmonella or botulinum toxin may be high, while the probability of cancer as a result of ingesting carcinogenic pesticides above the permissable levels may be much lower.

Finally, the value to consumers of avoiding the welfare losses associated with adverse health effects depends on how soon the effect occurs after they consume the violative product and the magnitude of the expected welfare losses.

This paper develops a methodology for estimating the value of the welfare gains associated with avoiding statistical cases of morbidity and mortality from foodborne illnesses. We demonstrate the methodology for botulism, salmonellosis, chronic hepatitis, and bladder cancer. The methods and results from this research can be combined with information on the costs of enforcement, dose-response relationships, and changing probabilities of violations to guide FDA in developing an efficient compliance monitoring program.

## II BACKGROUND

Consumers derive value from a food inspection and monitoring program through lower risks of adverse health effects. When a compliance monitoring program detects and removes a violative product from distribution, it reduces the risk of consumers suffering adverse health effects and corresponding welfare losses. The value of reducing the risks of adverse health effects could be easily measured by market clearing prices if there were markets for health risks. With the exception of wage premiums for occupations with higher than average risks of on-the-job death or injury, health risks are not a market commodity. Thus, analysts must develop other methods to estimate the value of reducing food-related health risks.

One of the earliest approaches used to estimate directly the costs associated with different illnesses is the cost-of-illness (COI) methodology. In its simplest form, the COI methodology calculates the dollar cost of illness or disease as the sum of the present values of the medical resources used to diagnose and treat the disease and the individual productivity losses it causes. The COI methodology is a practical simplification of the more comprehensive human capital approach to valuing illness. Cooper and Rice (1976) and Rice, Hodgson, and Kopstein (1985) have used the COI method to estimate the costs of many different diseases. Hartunian et al. (1981) employed the COI model to value the costs of coronary heart disease, stroke, cancer, and motor vehicle injuries.

The COI method is well-suited for estimating many of the tangible costs of illnesses, but it does not address any of the intangible or disutility costs. Nor does it distinguish between avoidance of identified cases of illness and reduction in the risk of adverse health effects. Utility is a conceptual device used primarily by economists to measure the amount of well-being and pleasure an individual experiences. Utility declines with deteriorating health status, as well as with increased risk of illness. Since the benefits of a government regulation are best described in terms of statistical cases of illness avoided, we first estimate the value of utility gains from decreased risks of statistical illness.

While utility is a useful construct in theory, it is unobservable in practice. Thus, we need to derive proxy measures for utility changes such as monetary values. The concept of willingness to pay (WTP) has gained acceptance in the economics profession as a dollar equivalent to utility changes. The WTP approach is based on microeconomic utility theory and has been used extensively to estimate the value of utility improvements and the cost of utility reductions. For example, the WTP approach imputes the cost of adverse health consequences by measuring how much individuals are willing to pay for small reductions in the risk of those effects. By measuring the value individuals place on small changes in the probability of mortality and morbidity, economists and health professionals have extended the analysis to measure the disutility cost of a statistical mortality and morbidity case.

Although dollars may be an imperfect measure of a consumer's valuation of avoided utility losses, within a certain range of preferences, people are familiar with the process of expressing values for goods and services through prices. Furthermore, dollar values provide a benchmark by which a wide variety of foodborne illnesses can be measured and compared.

We present a methodology for estimating the dollar value of avoiding morbidity and mortality from foodborne illnesses using both the willingness-to-pay approach and the cost of illness approach. We demonstrate our methodology and derive valuation estimates for avoiding statistical cases of botulism, salmonellosis, chronic hepatitis, and bladder cancer.

## III METHODS AND RESULTS

The empirical model presented here was developed using publicly available data. We used the model as part of a larger study to estimate the value of avoiding both health and nonhealth adverse effects from consuming foods that violate the FD&C Act (Mauskopf et al. 1988). In this paper, we only describe and implement the method for estimating the value of avoiding adverse health effects.

The method we use to compute the dollar value for avoiding foodborne illnesses associated with violations of the FD&C Act consists of the following seven steps and is illustrated in Figure 2:

- Identify the foodborne illnesses of concern.
- Describe the adverse health effects of each foodborne illness on an individual consumer.
- Translate these health effects into time spent in specific health states.
- Estimate the gains in quality-adjusted life-years (QALYs) associated with avoiding a case of each foodborne illness.
- Estimate the value of a QALY.
- Compute the willingness-to-pay estimate for avoiding each foodborne illness by combining the estimates of the QALYs avoided and the dollar value of a QALY.
- Use the estimated adverse health effects to compute the cost-of-illness estimates for each foodborne illness.

We discuss each step of the analysis in the following sections.

### Identify Foodborne Illnesses

In the first step of the analysis, we use available human and nonhuman data to identify illnesses likely to be associated with violations of the FD&C Act (FASEB, 1988). In some cases, a cause-and-effect relationship between a violation and an illness is well-established, such as that between botulinum toxin and botulism. In other cases, this relationship may be less understood, such as that between pesticide residues and risk of cancer.

To facilitate the later steps in the estimation procedure, we subdivide foodborne illnesses into three categories:

- acute illnesses, which occur with no latency period after exposure, have a well-defined duration, and end in either death or complete cure;
- chronic illnesses, which have no (or a short) latency period after exposure, a prolonged duration, and end in death; and

- cancers, which have a prolonged latency period, short or prolonged duration, and end in either death or complete cure.

Most foodborne illnesses can be assigned to one or more of these categories. Table I presents some examples of violations of the FD&C Act and their associated foodborne illnesses. Botulism is caused by botulinum toxin in a food product and is classified as an acute illness. Survivors of a severe case of botulism might also suffer from residual chronic illness, but this is not included in our analysis. Salmonellosis is caused by a bacterium and is a common disease in its less severe forms. Chronic hepatitis may persist throughout an individual's life after an attack of acute foodborne hepatitis. Certain pesticide residues and food coloring agents may be associated with an increased risk of bladder cancer.

### Describe the Health Impact on Consumers

In general, foodborne illnesses can occur at various levels of severity, each of which affects the consumer to a different degree. To simplify the analysis, we chose three levels of severity for each illness: mild, moderate, and severe. We define the severity category for the acute and chronic illnesses based on well-defined clusters of symptoms, resource use, and/or mortality risk. The severity levels are used for all illnesses except cancers, which we define as local, regional, and distant.

For each level of illness severity, we describe the impact on consumers in terms of patient symptoms, mortality rates, duration of treatment and recovery, frequently used medical treatment, and functional status during treatment and recovery. Functional status during the illness is defined as either in a hospital, in bed at home, or at home not in bed. Table II illustrates an impact profile for botulism, salmonellosis, and chronic hepatitis. Table III illustrates the impact profile for bladder cancer. We obtained the data for these impact descriptions from the medical and clinical literature.

### Determine Time Spent in Specific Health States

Adverse health effects from foodborne illnesses can cause both short- and long-term changes in health status. We classify the length and degree of health status changes by the time spent in specific health states. Health states can be defined broadly or narrowly depending on the conditions and purpose of the analysis. Several studies in the biomedical literature have developed health states or health status index scales to describe and categorize the adverse health consequences from illness and disease.

For this analysis, we use the set of health states defined by Rosser and Kind (1978). But analysts can use any set of health states general enough to be applied to all foodborne illnesses and for which relative utility weights have been estimated. In our comprehensive study for FDA (Mauskopf et al. 1988), we also used the Bush et al. (1981) health status index and the health status index developed for a study of vaccines by the Institute of Medicine (1985). Table IV presents the Rosser and Kind health state definitions. They express health status in terms of two dimensions: objective disability and distress.

After choosing a set of health states, we describe the adverse health effects from each foodborne illness in terms of time spent in the specific health states. The descriptions are presented for botulism, salmonellosis, and chronic hepatitis in Table V and for bladder cancer in Table VI using the Rosser and Kind health states. For example, we estimated that a mild case of botulism would result in severely limited ability to work for five days with mild distress. In contrast, we estimated a serious case of botulism would result in 90 days confined to bed in severe distress, 30 days confined to a chair in moderate distress, and 60 days unable to work in mild distress.

### Estimate Losses in Quality-Adjusted Life-Years

To estimate the QALYs lost as a result of a foodborne illness, it is necessary to make a series of assumptions including age at exposure to the violative product, latency period after exposure for the illness to appear, remaining life expectancy at time of illness, and health status at onset of illness and for remaining lifetime. We assume the following baseline conditions:

- age at exposure is 30 years,
- a 20-year latency period for cancer, but no latency period for acute or chronic effects,
- remaining life expectancy at age 30 and at age 50 is 46 years and 26 years respectively,
- individuals are in perfect health and, in the absence of foodborne illness, would continue in perfect health for their remaining lifetime.

Lipscomb et al. (1983) have shown that this last assumption results in overestimates of the losses associated with illness of about 5 percent.

Using the assumptions noted above, the estimated time spent in specific health states for each foodborne illness, and the relative utility (well-being) weights shown in Table VII for the Rosser and Kind index, we computed the losses in QALYs associated with each illness.

Table VIII presents the estimated losses in QALYs for botulism, salmonellosis, chronic hepatitis, and bladder cancer.

For botulism, the estimated losses in QALYs are much larger for those who die from the disease (25.5 QALYs discounted at 3 percent or 46 QALYs undiscounted) than for those who have a severe case and survive (0.647 QALYs). For chronic hepatitis, the losses in QALYs are assumed to continue for the rest of the individual's lifetime. We estimate that approximately 50 percent of the people with bladder cancer die. In addition to suffering premature death, those individuals who die from bladder cancer suffer significantly greater losses from morbidity (0.31 undiscounted QALYs) than those who survive (0.07 undiscounted QALYs).

### Estimate the Value of a Quality-Adjusted Life-Year

We use willingness-to-pay estimates for reductions in morbidity and mortality risks to assign a dollar value to a QALY. The process follows a series of steps. First, we explored the literature and chose a representative willingness-to-pay estimate for the value of a statistical life. We selected $5 million. This value was estimated by Viscusi and Moore (1988) in a recent study of wage premiums paid to workers in risky occupations with an average age of 40 years. Five million dollars serves as the willingness-to-pay estimate to avoid the index state (death) from a previous condition of perfect health. We assume that the remaining life expectancy for a 40-year-old worker is 36 years. Using a value estimated for a statistical life (death) is appropriate, because FDA monitors and enforces programs that reduce the risk of foodborne illness for the general population, thus preventing statistical, not identified, cases.

Equation 1 illustrates the formula we use to compute the undiscounted value of a QALY from the estimated value of a statistical life.

$$\text{\$QALY (0\% discount)} = \frac{\text{value of a statistical life}}{\text{remaining life expectancy}} \qquad (1)$$

Alternatively, for a discount rate of 3 percent, we first convert remaining life expectancy to total discounted life-years (TDLYs) through the following calculation:

$$\text{TDLYs remaining} = \sum_{i=1}^{36} \frac{1}{(1+0.03)^{i-1}} = 22.5, \qquad (2)$$

and then compute the value of a QALY as:

$$\text{\$QALY (3\% discount)} = \frac{\text{value of a statistical life}}{\text{total discounted life-years}} \qquad (3)$$

Using $5,000,000 as the value of a statistical life (Viscusi and Moore, 1988), the estimated value of a QALY is $138,000 at a 0 percent discount rate, and $222,222 at a 3 percent discount rate.

In computing the value of a QALY as described above, we used death as the index state. Alternatively, the value of a QALY can be computed from estimates of the willingness-to-pay to avoid other adverse health states, provided that the lost QALYs associated with these adverse health effects are also estimated. For example, Rowe and Chestnut (1984) estimated the willingness to pay to avoid a bad asthma day at $23.00. Using the Rosser and Kind scale, the loss in QALYs associated with a day with asthma is estimated as 0.00008. Thus, using a day of asthma as the index state will result in an estimate for a QALY of $287,500. This exercise can be performed for a variety of different index states to generate a range of estimates for the value of a QALY.

## Estimate the Value of Avoiding Morbidity and Mortality

In the final step of the willingness-to-pay analysis, we compute the product of the QALYs gained and the dollar value of a QALY to generate willingness to-pay estimates for the avoided morbidity and mortality associated with foodborne illnesses. Estimated values for botulism, salmonellosis, chronic hepatitis, and bladder cancer are presented in Table IX. The estimated dollar value for avoiding foodborne illnesses associated with a high risk of death, such as severe botulism or bladder cancer, is much higher than for avoiding nonfatal illnesses such as mild or moderate cases of salmonellosis. Nevertheless, the estimated morbidity losses are not insignificant.

Caution must be exercised when interpreting the implications of these estimates. Many serious foodborne illnesses are rare, such as those presented as examples here. Since the willingness-to-pay values are for statistical cases of each illness, the aggregate value of avoiding all cases may be relatively small in comparison to a less severe illness with a much higher prevalence. As an example, foodborne illnesses such as salmonellosis are usually not life threatening, yet they are very common, especially in their milder forms. Consequently, the total dollar losses associated with morbidity from this disease may be very high—in the billions of dollars (Archer and Kvenberg 1985).

## Estimate Morbidity and Mortality Losses Using the Cost-of-Illness Approach

An alternative approach to estimating the value of avoiding foodborne illnesses is to estimate the direct and indirect costs avoided in terms of medical care and productivity losses.

The cost-of-illness method advocates an accounting cost framework to estimate the observable costs (medical care) and an opportunity cost framework to estimate the implicit costs (productivity losses). Cost-of-illness estimates for botulism, salmonellosis, and bladder cancer are presented in Table X.

Cost-of-illness methods have been applied in numerous studies for many different illnesses and diseases. Despite its popularity, the cost-of-illness method tends to underestimate the true value of the avoided illness because it does not address the value of avoiding certain cost categories (e.g., pain and suffering). On the other hand, the cost-of-illness method may overestimate the value of the avoided medical costs to the individual because these costs are often shared via health insurance.

## IV CONCLUSION

We described two methods that can be used to estimate the value of avoiding the morbidity and mortality associated with foodborne illnesses: willingness-to-pay and cost-of-illness. We demonstrated the use of these methods and estimated the value of avoiding statistical cases of four foodborne illnesses: botulism, salmonellosis, chronic hepatitis, and bladder cancer. At least three conclusions can be drawn as a result of this analysis. First, the fatality rate is the key factor when determining the relative value of avoiding different levels of severity for acute illnesses and cancers. Second, the value of morbidity losses, both for those ultimately dying from the illness and for those surviving, are significant. Finally, the estimated value of avoiding chronic diseases is critically dependent on the degree of functional impairment associated with the illness.

Although the cost-of-illness method is a convenient approach for estimating the tangible costs of illness and disease, it is flawed because it does not consider disutility costs. Willingness-to-pay methods are conceptually appealing because they are based on microeconomic utility theory. Willingness-to-pay estimates include the disutility costs associated with illness and disease such as physical and emotional pain and suffering.

Despite its theoretical strengths, the willingness-to-pay approach can be difficult to implement due to data requirements. In addition, the estimates are highly sensitive to simplifying assumptions and baseline parameter values (e.g., age at exposure, remaining life expectancy, discount rate, health status index scale). Although these issues cannot be ignored, our methodology is able to use secondary data to generate defensible estimates for the value of avoiding a wide variety of morbidity states. More importantly, decisionmakers can use this

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methodology to include the value of reducing morbidity risks as well as the value of reducing mortality risks in their benefits estimates. This is especially useful for FDA and other federal agencies that regulate health risks.

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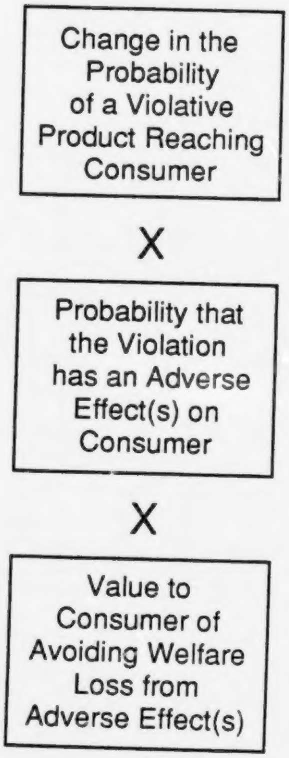
Change in the Probability of a Violative Product Reaching Consumer
X
Probability that the Violation has an Adverse Effect(s) on Consumer
X
Value to Consumer of Avoiding Welfare Loss from Adverse Effect(s)

**Figure 1. Benefits of Compliance Monitoring**

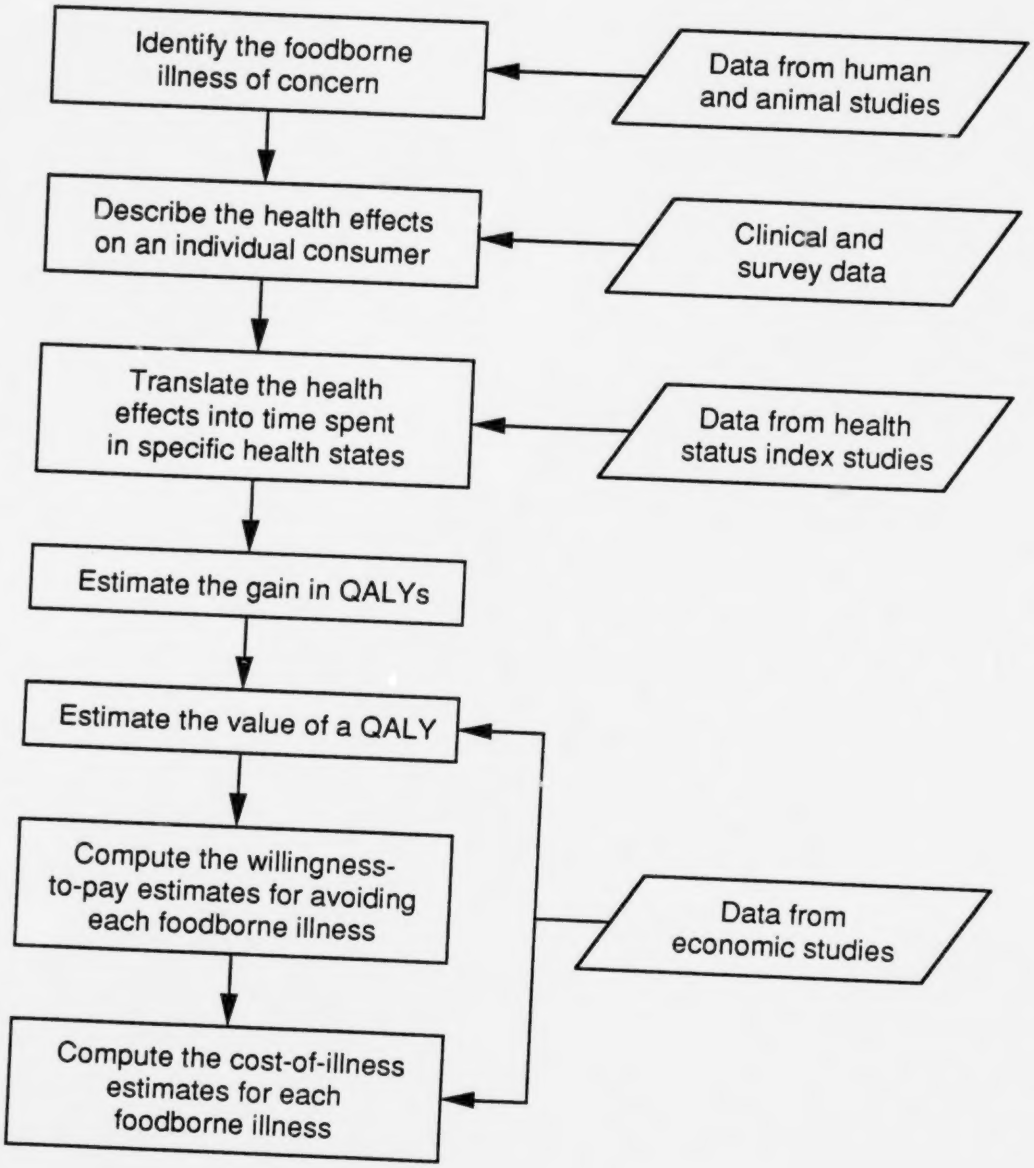
Identify the foodborne illness of concern
Data from human and animal studies
Describe the health effects on an individual consumer
Clinical and survey data
Translate the health effects into time spent in specific health states
Data from health status index studies
Estimate the gain in QALYs
Estimate the value of a QALY
Compute the willingness-to-pay estimates for avoiding each foodborne illness
Data from economic studies
Compute the cost-of-illness estimates for each foodborne illness

Figure 2. Flow Diagram of Estimation Model

## TABLE I. SAMPLE OF FOODBORNE ILLNESSES CAUSED BY VIOLATIONS OF THE FD&C ACT

| Violation | Acute Effects | Chronic Effects | Cancers |
|---|---|---|---|
| *FD & C Red #10* | Contact dermatitis | | Bladder |
| *Cat filth/damage* | Toxoplasmosis | Congenital toxoplasmosis | |
| *C. Botulinum* | Botulism | | |
| *Human filth* | Shigellosis, hepatitis, listeriosis, colitis | Chronic hepatitis, cirrhosis | Liver |
| *Salmonella* | Salmonellosis | | |
| *Inadequate pasteurization, LACF* | Salmonellosis, botulism | | |
| *Sulfite* | Allergic response | | |

### TABLE II. HEALTH EFFECTS OF BOTULISM, SALMONELLOSIS, AND CHRONIC HEPATITIS

| Illness | Symptoms | Duration | Treatment | Functional Status | Fatality Rate |
|---|---|---|---|---|---|
| **Botulism** | | | | | |
| *Mild* | Malaise, weakness, fatigue | 5 days | Antitoxin | 5 house days | 0% |
| *Moderate* | Nausea/vomiting, diarrhea, abdominal pain, fever, malaise, weakness, headache, dizziness | 21 days | Antitoxin | 7 hospital days<br>7 bed days<br>7 house days | 0% |
| *Severe* | Same as moderate plus respiratory paralysis, muscular paralysis, pulmonary infection | 180 days | Antitoxin, respiratory support | 90 hospital days<br>30 bed days<br>60 house days | 22.5% |
| **Salmonellosis** | | | | | |
| *Mild* | Nausea/vomiting, diarrhea, abdominal pain, anorexia, weakness | 3 days | Oral fluids, antispasmodics | 2 bed days<br>1 house day | 0% |
| *Moderate* | Same as mild plus fever, headache, dehydration/ prostration | 7 days | Oral fluids, antispasmodics | 4 bed days<br>3 house days | 0% |
| *Severe* | Same as moderate plus enteric bacteremia | 11-20 days | I.V. fluids, antispasmodics, antinbiotics | 5-14 hospital days<br>3 bed days<br>3 house days | 13% |
| **Chronic Hepatitis** | | | | | |
| | Malaise | 1 year to lifetime | None | Very minor restrictions | 0% |

Sources: FASEB (1988), Mann et. al., (1983), Todd (1985a), Todd (1985b), CDC (1980).

## TABLE III. HEALTH EFFECTS OF BLADDER CANCER

Estimated Duration of Treatment, Cured Patients = < 2 years

Estimated Duration of Treatment, Uncured Patients = 1.97 years

Estimated Fatality Rate = 51%

### Associated Signs and Symptoms

*Bloody urine*
*Pain on urinating*
*Abdominal pain*
*Further symptoms from metastases*

### Frequently Used Medical Treatments and Associated Side Effects

- Surgery
  - *Pain*
  - *Discomfort*
- Radiation Therapy
  - *Diarrhea*
  - *Mucositis which can preclude substantial oral intake and lead to malnutrition*
- Chemotherapy
  - *Nausea*
  - *Vomiting*
  - *Hair loss*
  - *Inflammation of mucous membranes*
  - *Suppression of white cell development*
  - *Cerebellar dysfunction at high doses*
  - *Anorexia*
  - *Rashes*
  - *Inflammation of hair follicles*
  - *Hyperpigmentation*
  - *Fever/chills*
  - *Renal failure*
  - *Anemia*

### Functional Status During Treatment and Recovery

| **Cured Patients** | **First Year** | **Second Year** |
|---|---|---|
| Hospital Days | 10 | 7 |
| Days of Hospital Recovery | 8 | 6 |
| Chemotherapy Days | 0 | 0 |
| Days of Chemotherapy Recovery | 0 | 0 |
| Radiation Therapy Days | 1 | 0 |
| Days of Radiation Therapy Recovery | 1 | 0 |
| Mild Distress Days | 345 | 170 |

| **Uncured Patients** | **First Year** | **Second Year** |
|---|---|---|
| Hospital Days | 18 | 35 |
| Days of Hospital Recovery | 14 | 28 |
| Chemotherapy Days | 3 | 24 |
| Days of Chemotherapy Recovery | 3 | 24 |
| Radiation Therapy Days | 7 | 14 |
| Days of Radiation Therapy Recovery | 3 | 7 |
| Nursing Home Days | 0 | 7 |
| Partial Disability Days | 0 | 41 |
| Total Disability Days | 0 | 41 |
| Mild Distress Days | 317 | 144 |

## TABLE IV. ROSSER AND KIND HEALTH STATES

| Objective Disability | Distress |
|---|---|
| 1. None | 1. None |
| 2. Slight social disability | 2. Mild |
| 3. Severe social disability, slight impairment at work | 3. Moderate |
| 4. Work severely limited | 4. Severe |
| 5. Unable to work | |
| 6. Confined to chair | |
| 7. Confined to bed | |
| 8. Unconscious | |

Source: Rosser and Kind (1978)

## TABLE V. DISABILITY, DISTRESS, AND TIME IN SPECIFIC HEALTH STATES FOR BOTULISM, SALMONELLOSIS, AND CHRONIC HEPATITIS

| Illness | Disability Index | Distress Index | Duration |
|---|---|---|---|
| **Botulism** | | | |
| *Mild* | 4 | 2 | 5 days |
| *Moderate* | 7 | 3 | 7 days |
| | 6 | 3 | 7 days |
| | 4 | 2 | 7 days |
| *Severe* | 7 | 4 | 90 days |
| | 6 | 3 | 30 days |
| | 4 | 2 | 60 days |
| **Salmonellosis** | | | |
| *Mild* | 6 | 3 | 1 days |
| | 4 | 2 | 1 days |
| *Moderate* | 6 | 3 | 4 days |
| | 4 | 2 | 3 days |
| *Severe* | 7 | 3 | 10 days |
| | 6 | 3 | 3 days |
| | 4 | 2 | 3 days |
| **Chronic Hepatitis** | 2 | 2 | 365 days/year |

## TABLE VI. DISABILITY, DISTRESS, AND TIME IN SPECIFIC HEALTH STATES FOR BLADDER CANCER

| Duration | | | Functional Status During Treatment and Recovery* | |
|---|---|---|---|---|
| First Year | Second Year | | Disability Index | Distress Index |
| | | **Cured Patients** | | |
| 10 | 7 | Hospital Days | 7 | 3 |
| 8 | 6 | Days of Hospital Recovery | 6 | 3 |
| 0 | 0 | Chemotherapy Days | 5 | 3 |
| 0 | 0 | Days of Chemotherapy Recovery | 4 | 2 |
| 1 | 0 | Radiation Days | 5 | 3 |
| 1 | 0 | Days of Radiation Recovery | 4 | 2 |
| 345 | 170 | Mild Distress Days | 1 | 2 |
| | | **Uncured Patients** | | |
| 18 | 35 | Hospital Days | 7 | 3 |
| 14 | 28 | Days of Hospital Recovery | 6 | 3 |
| 3 | 24 | Chemotherapy Days | 6 | 3 |
| 3 | 24 | Days of Chemotherapy Recovery | 5 | 3 |
| 7 | 14 | Radiation Days | 6 | 3 |
| 3 | 7 | Days of Radiation Recovery | 5 | 3 |
| 0 | 7 | Nursing Home Days | 7 | 3 |
| 0 | 41 | Partial Disability Days | 4 | 2 |
| 0 | 41 | Total Disability Days | 6 | 3 |
| 317 | 144 | Mild Distress Days | 1 | 2 |

* Weighted average for cases diagnosed in local, regional, and distant stages.

## TABLE VII. RELATIVE UTILITY WEIGHTS FOR THE ROSSER AND KIND HEALTH STATUS INDEX

| Disability Index | Distress Index | | | |
|---|---|---|---|---|
| | *1* | *2* | *3* | *4* |
| *1* | 1.0 | 0.995 | 0.990 | 0.967 |
| *2* | 0.990 | 0.986 | 0.973 | 0.932 |
| *3* | 0.980 | 0.972 | 0.956 | 0.912 |
| *4* | 0.964 | 0.956 | 0.942 | 0.870 |
| *5* | 0.946 | 0.935 | 0.900 | 0.700 |
| *6* | 0.875 | 0.845 | 0.680 | 0.000 |
| *7* | 0.677 | 0.564 | 0.000 | -1.486 |
| *8* | -1.028 | — | — | — |

Source: Rosser and Kind (1978)

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## TABLE VIII. LOSSES IN QUALITY-ADJUSTED LIFE-YEARS FROM BOTULISM, SALMONELLOSIS, CHRONIC HEPATITIS, AND BLADDER CANCER

| Illness | Fatality Rate | Loss for Survivors QALYs* (QALDs)** | Weighted Average Loss QALYs* (QALDs)** |
|---|---|---|---|
| **Botulism** | | | |
| *Mild* | 0% | 0.00055<br>(0.2) | 0.00055<br>(0.2) |
| *Moderate* | 0% | 0.0263<br>(9.6) | 0.0263<br>(9.6) |
| *Severe* | 22.5% | 0.647<br>(236) | 6.24<br>(2,279) |
| **Salmonellosis** | | | |
| *Mild* | 0% | 0.001<br>(0.4) | 0.001<br>(0.4) |
| *Moderate* | 0% | 0.004<br>(1.4) | 0.004<br>(1.4) |
| *Severe* | 13% | 0.03<br>(11.1) | 3.35<br>(1,221) |
| **Chronic Hepatitis** | 0% | 0.36<br>(130.4) | 0.36<br>(130.4) |
| **Bladder Cancer** | | | |
| *Undiscounted* | 51% | 0.068<br>(24.7) | 12.9<br>(4,700) |
| *Discounted 3% to Diagnosis* | 51% | 0.067<br>(24.4) | 9.57<br>(3,494) |
| *Discounted 3% to Exposure* | 51% | 0.037<br>(13.5) | 5.30<br>(1,934) |

* QALY = quality-adjusted life-year
** QALD = quality-adjusted life-day

## TABLE IX. WILLINGNESS-TO-PAY ESTIMATES FOR AVOIDING BOTULISM, SALMONELLOSIS, CHRONIC HEPATITIS, AND BLADDER CANCER

| Illness | Fatality Rate | Survivors | Weighted Average |
|---|---|---|---|
| **Botulism** | | | |
| *Mild* | 0% | $130 | $130 |
| *Moderate* | 0% | $5,800 | $5,800 |
| *Severe* | 22.5% | $143,750 | $1,388,000 |
| **Salmonellosis** | | | |
| *Mild* | 0% | $222 | $222 |
| *Moderate* | 0% | $890 | $890 |
| *Severe* | 13% | $6,700 | $740,000 |
| **Chronic Hepatitis** | 0% | $79,400 | $79,400 |
| **Bladder Cancer** | | | |
| *Undiscounted* | 51% | $8,220 | $1,178,000 |
| *Discounted 3% to Diagnosis* | 51% | $9,384 | $1,780,000 |
| *Discounted 3% to Exposure* | 51% | $14,900 | $2,127,000 |

**TABLE X. COST-OF-ILLNESS ESTIMATES FOR AVOIDING BOTULISM, SALMONELLOSIS, AND BLADDER CANCER**

| Illness | Fatality Rate | Survivors | Weighted Average |
|---|---|---|---|
| **Botulism** | | | |
| *Mild* | 0% | $470 | $470 |
| *Moderate* | 0% | $4,710 | $4,710 |
| *Severe* | 22.5% | $68,500 | $195,000 |
| **Salmonellosis** | | | |
| *Mild* | 0% | $197 | $197 |
| *Moderate* | 0% | $622 | $622 |
| *Severe* | 13% | $65,556 | $86,895 |
| **Bladder Cancer*** | 51% | $13,876 | $215,000 |

* Lost earnings discounted at 3% to diagnosis.

# UTILITY-ADJUSTED IMPAIRMENT YEARS: A LOW-COST APPROACH TO MORBIDITY VALUATION

by

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## Introduction

This article extends the Arthur (1981) social consumption equivalent (SCE) value of life model to one that also accounts for health status and serious injury. Death is only one possible outcome of risky activities, and by the available evidence, not always the least desirable. Fates worse than death are now recognized as important determinants of private decisions to avoid risk and of the social value of public programs designed to reduce or eliminate hazards to life and limb. Kind, Rosser, and Williams (1982) examined the impacts of distress and disability on the joy of living and found that permanent confinement to bed was considered as bad as death, and permanent coma even worse. In a British study of injury severity by Green and Brown (1978), university students ranked death third behind brain damage and paralysis from the neck down. Jones-Lee, Hammerton, and Philips (1985) found that in a probability sample of 1000 British residents, the median individual considered lifetime confinement to a wheelchair as bad as death, and being permanently bedridden was considered as bad or worse than death by 63 percent of the respondents. Howard (1984) has examined the theoretical implications of extreme disability for individual decisions regarding risk.

The impact of serious injury on individual and social welfare can be substantial, as implied by the findings cited above. Implicit in these data is the effect of injuries on the utility from additional years of life. A person's health status is likely to have a direct effect on welfare--particularly when pain and suffering are involved--as well as indirect effects such as diminished utility from consuming other goods.

In addition to their effects on the utility associated with additional years of life, permanent and temporary disabilities have important implications for the age profile of consumption, production, and mortality. Changes in the incidence of serious illness and injury also may have quite different implications than changes in the death rate from the same cause. The impact of a change in the incidence of serious injuries on labor market productivity and consumption may include offsetting effects, for example, depending on whether the change is associated with an increase or decrease in death rates. Reductions in the injury rate that are not offset by an increase in death rates should increase average labor productivity. The magnitude of these effects will depend on the age of the individual, time to recovery, and the extent to which it was already possible to switch to less physically demanding activities following a serious injury. Consumption of costly medical resources will decline with a reduction in the incidence of serious injury, perhaps more than offsetting any increase in other types of consumption.

The mortality implications of adding the seriously injured to the model can be viewed in terms of resuscitated lives--saving those who would have died as the result of a serious illness or injury through the application of advanced medical technology or improved health and safety measures--and should be contrasted with the elimination of a cause of death. The life-table implications of lifesaving of this type have been worked out in detail by Vaupel and Yashin (1985). Those who have been saved from death but not from serious injury subsequently face a different regime of mortality risks than those who have never been seriously injured. For example, the quadraplegic must forgo risk-producing activities, such as driving, but faces increased risks to life in other respects, for example, from infections.

The social consumption equivalent (SCE) framework allows one to trace the implication of changes in mortality across different ages on the various components of the model. The following section briefly summarizes the main points of the SCE model as it has been developed for death. Many of the more technical details are included in a footnote. The discussion includes a comparison of the SCE model with those based on willingness to pay and human capital. This is followed by a formal presentation of the revised model that includes health status as an additional argument of the utility function and a determinant of the age patterns of productivity, consumption, and mortality. Once health statuses are refined beyond the simple two-way classification alive-dead, it is necessary to confront the problem of measuring the utilities of alternative health states. The paper then reviews the utility measures available and examines their consistency and capability through illustrative valuations of selected illnesses and injuries.

## The Social Consumption Equivalent Value of Life

The SCE method uses an age-specific, overlapping generation, economic model to assess the cost of loss of life or the value of lives saved as the result of a change in the pattern of mortality by age. The SCE method is: (1) based on economic welfare theory, (2) gives values in dollar terms that are a function of the age of the victim, (3) gives values that can be expressed in terms of human capital and willingness to pay, and (4) is fully actuarial. Under SCE, loss of life can be evaluated in three different ways: (1) by changes in age-specific life-table survival risks (caused, say, by improved highway design), (2) by "statistical" lives lost at a given age a, and (3) by cause (cancer, airline accidents) where loss of life occurs with a known age incidence. SCE emphasizes that valuation must account for the additional consumption of those whose lives are saved or lengthened. For example, when a 70 year-old's life is "saved," society gains that person's enjoyment or utility of additional years that are otherwise lost. But the extra consumption that supports utility in these additional years must be paid for--possibly by additional social security payments, by transfers from younger relatives, or by additional saving earlier in life.

The SCE method can be viewed in terms of two key relationships. The first of these is the social welfare function given by:

$$W = \int_0^{\omega} U[c(x),x]p(x)dx \tag{1}$$

where U[c,x] is the utility of being alive at age x, given consumption rate c; p(x) is the probability of surviving from birth to age x; and ω is the maximum age of survivorship. The second equation is given by the societal budget constraint:

$$\int_0^{\omega} e^{-gx}p(x)c(x)dx = (f(k)-gk) \int_0^{\omega} e^{-gx}p(x)\lambda(x)dx \tag{2}$$

where g is the constant rate of population growth; $f(k)=F(K,L)/L$ is output per worker at capital-labor ratio K/L for an economy with constant returns to scale production function F; and λ(x) is the age schedule of labor participation.[1]

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By considering the total differentials of equations (1) and (2) with respect to an arbitrary pattern of changes in survival probabilities, Arthur was able to show that the change in expected lifetime welfare is given by:

$$\delta W = \int_0^\omega U[c(x),x]\delta p(x)dx$$

$$+ \partial U/\partial c(0)\ \{w\int_0^\omega e^{-gx}\lambda(x)\delta p(x)dx - \int_0^\omega e^{-gx}c(x)\delta p(x)dx + \beta\delta g[\delta p]\ \} \qquad (3)$$

where w is the wage rate, and $\beta$ is the life-cycle value of a marginal increase in the population growth rate (Arthur and McNicoll, 1978). This can be reexpressed more conveniently as:

$$\underbrace{\delta W}_{\substack{\text{Life-Cycle}\\ \text{Welfare}\\ \text{Increase}}} = \underbrace{U_{\delta p}}_{\substack{\text{Utility of}\\ \text{Extra}\\ \text{Life-Years}}} + \partial U/\partial c(0)\ \{\ \underbrace{wL_{\delta p}}_{\substack{\text{Value of}\\ \text{Extra}\\ \text{Labor-Years}}} - \underbrace{c_{\delta p}}_{\substack{\text{Social Cost of}\\ \text{Consumption}\\ \text{Upkeep}}} + \underbrace{v_{\delta p}\beta/A_m}_{\substack{\text{Value of}\\ \text{Additional}\\ \text{Children}}}\ \} \qquad (4)$$

where $L_{\delta p}$ and $c_{\delta p}$ are the expectations of extra person-years of production and consumption resulting from the particular variation $\delta p$, $v_{\delta p}$ is additional children per person due to the variation in mortality, and $A_m$ is the average age of reproduction in the stable population.

Equation (4) was used by Arthur (1981) to develop the social welfare equivalent of an increase in risk correponding to the loss of one life selected at random at age a. In order to express the value of life in consumption units, Arthur assumed that utility function U does not vary with age and has constant elasticity of consumption:

$$U[c(x),x] = c^{\varepsilon}$$

where $\varepsilon=(dU/dc)(c/U(c))$ is the constant elasticity of consumption. (5) The social consumption equivalent value of mortality variation $\delta p$ is given by:

$$SCE[\delta p] = (1/\varepsilon - 1)c_{\delta p} + wL_{\delta p} + (\beta/A_m)v_{\delta p}. \qquad (6)$$

Equation (6) makes explicit the fact that the enjoyment of additional years of life is directly offset by its consumption cost.

Equation (6) also provides a direct connection between the social consumption equivalent method and human capital and willingness-to-pay criteria for valuing variations in mortality risks. In Landefeld and Seskin's (1982) formulation, willingness to pay for life and safety (WTP) essentially equals the product of the present value of the individual's future monetary and nonmonetary goods consumption times A, the reciprocal of the goods consumption elasticity of lifetime utility. The first component is given in equation (6) by $c_{\delta p}$, and the second by $1/\varepsilon$. The human capital (HK) approach uses the present value of the change in expected lifetime earnings, which appears as the $wL_{\delta p}$ term in equation (6). Human capital net of consumption, thus, equals the negative part of the first term in (6) plus the second term. This suggests the following definition of SCE in terms of WTP and HK:

$$\underbrace{SCE[\delta p]}_{\substack{\text{Social}\\ \text{Consumption}\\ \text{Equivalent}\\ \text{Value}}} = \underbrace{(1/\varepsilon)c_{\delta p}}_{\substack{\text{Average}\\ \text{Individual}\\ \text{Willingness}\\ \text{to Pay}}} + \underbrace{wL_{\delta p} - c_{\delta p}}_{\substack{\text{Human Capital}\\ \text{Net of}\\ \text{Consumption}}} + \underbrace{(\beta/A_m)v_{\delta p}}_{\substack{\text{Value of}\\ \text{Additional}\\ \text{Children}}}. \qquad (7)$$

Equation (7) shows that society's willingness to pay for mortality improvements may be greater or less than individual willingess to pay for the same change.

## Adding Health Status to the Model

The SCE model can modified to include nonfatal risks by including a term for health status in the welfare function. We assume that each person has a utility function $U[c(x),h(x),x]$, where $h(x)$ is defined as the "state of health" at age x. Health status is also assumed to have a direct impact on health costs, consumption, fertility, mortality, and labor productivity. Changes in fertility, mortality, and labor productivity will induce changes in the equilibrium stable population growth rate and the equilibrium capital-labor ratio. Suppose that some activity (e.g., less safe roads, changed airline regulations) alters the health state by $\delta h(x)$ over the age dimension. Suppose also that this change has associated with it direct health costs $\delta c_H[\delta h]$, and alterations in consumption $\delta c[\delta h]$, labor effectiveness $\delta\lambda[\delta h]$, mortality $\delta p[\delta h]$, and fertility $\delta m[\delta h]$. The latter are all directly observed changes for a specific category of injuries.

The social welfare function now takes the following form:

$$W = \int_0^{\omega} U[c(x),h(x),x]\cdot p(x)dx. \qquad (8)$$

We can rewrite the societal budget constraint as:

$$\int_0^{\omega} e^{-gx}p(x)c(x)dx + \int_0^{\omega} e^{-gx}p(x)c_H(x)dx \equiv (f(k)-gk)\int_0^{\omega} e^{-gx}p(x)\lambda(x)dx \qquad (9)$$

breaking out health costs and consumption expenses separately. The change in welfare caused by $\delta h$ is given by:

$$\delta W = \partial U/\partial c(0) \int_0^\omega e^{-gx}\delta c[\delta h]p(x)dx + \int_0^\omega \partial U/\partial h \cdot \delta h(x)p(x)dx$$

Welfare Change
of Adjustment in
Consumption

Welfare Change Due to
Changed Incidence of
Injuries

$$+ \int_0^\omega U[c(x),h(x),x]\delta p[\delta h]dx. \qquad (10)$$

Welfare Change from
Extra Years of Life

The change in h will also cause adjustments across the societal budget constraint:

$$\begin{aligned} 0 = &\int_0^\omega e^{-gx}c(x)\delta p[\delta h]dx + \int_0^\omega e^{-gx}\delta c[\delta h]p(x)dx \\ &+ \int_0^\omega e^{-gx}c_H(x)\delta p[\delta h]dx + \int_0^\omega e^{-gx}\delta c_H[\delta h]p(x)dx \\ &- (f(k)-gk)\ \{ \int_0^\omega e^{-gx}\lambda(x)\delta p[\delta h]dx + \int_0^\omega e^{-gx}p(x)\delta\lambda[\delta h]dx\ \} \\ &- \delta k[\delta h](f'-g) \int_0^\omega e^{-gx}\lambda(x)p(x)dx - \beta\delta g[\delta h]. \end{aligned} \qquad (11)$$

Equation (11) is identical to equation (N.6) in footnote 1 except for the addition of the terms related to changes in medical costs ($\delta c_H[\delta h]$) and changes in labor productivity related to changes in health status ($\delta\lambda[\delta h]$). Also, the change in the population growth rate now includes the combined effect of changes in fertility and mortality.

Using equation (11) to substitute for the first term in equation (10) yields:

$$\delta W = \int_0^\omega U[c(x),h(x),x]\delta p[\delta h]dx + \int_0^\omega \partial U/\partial h\cdot\delta h(x)p(x)dx$$
$$+ \partial U/\partial c(0)\ \{\ w\cdot[\ \int_0^\omega e^{-gx}\lambda(x)\delta p(x)dx - \int_0^\omega e^{-gx}\delta\lambda[\delta h]p(x)dx\ ]$$
$$- \int_0^\omega e^{-gx}\delta c_H[\delta h]p(x)dx - \int_0^\omega e^{-gx}c_H(x)\delta p(x)dx$$
$$- \int_0^\omega e^{-gx}c(x)\delta p(x)dx + \beta\delta g[\delta h]\ \} \qquad (12)$$

This can be simplified to:

$$\underset{\substack{\text{Life-Cycle}\\ \text{Welfare}\\ \text{Increase}}}{\delta W} = \underset{\substack{\text{Utility of}\\ \text{Extra}\\ \text{Life Years}}}{U_{\delta p}} + \underset{\substack{\text{Utility of}\\ \text{Improved}\\ \text{Health Status}}}{U_{\delta h}}$$
$$+ \partial U/\partial c(0)\ \{\ \underset{\substack{\text{Value of}\\ \text{Extra}\\ \text{Labor Years}}}{wL_{\delta p}} - \underset{\substack{\text{Value of}\\ \text{Increased}\\ \text{Productivity}}}{wL_{\delta h}}$$
$$- \underset{\substack{\text{Social Cost of}\\ \text{Health Status}\\ \text{Improvements}}}{c_{H,\delta h}} - \underset{\substack{\text{Social Cost of}\\ \text{Health Maintenance}\\ \text{Over Extra Years}}}{c_{H,\delta p}}$$
$$- \underset{\substack{\text{Social Cost of}\\ \text{Consumption}\\ \text{Upkeep}}}{c_{\delta p}} + \underset{\substack{\text{Value of}\\ \text{Additional}\\ \text{Children}}}{(v_{\delta p}+v_{\delta m})\beta/A_m}\ \} \qquad (13)$$

where $L_{\delta p}$, $c_{\delta p}$, and $c_{H,\delta p}$ are expected extra person-years of production, consumption, and health costs, respectively, resulting from variation in mortality; $L_{\delta h}$, $c_{\delta h}$, and $c_{H,\delta h}$ are the expected life-cycle increases in productivity, consumption, and health costs directly associated with improved health status; $v_{\delta p}$ and $v_{\delta h}$ are additional children per person due to variation in mortality and health, respectively; and $A_m$ is the average age of reproduction in the stable population.

A comparison of equation (13) with equation (3) indicates that improving health status has benefits and cost above and beyond those associated with improved longevity. There is a quality-of-life aspect to living longer, now captured by the second term in equation (13), that was ignored in the original model. A healthier population will also be a more productive one, but at the additional social cost of maintaining good health. Finally, health status changes may affect fertility rates, which in turn affect social welfare either negatively or positively depending on the value of additional children to the society.

## Empirical Estimation

This subsection describes methods for estimating each term in equation (13). The remainder of this section provides illustrative applications.

Since health status is accounted for explicitly in the model, the utility per life year (the first term in the equation) should be uniform over time. Its value can be estimated from a study of individual willingness to pay for a statistical life by (a) selecting a discount rate, (b) computing the present value (in years) of the remaining expected lifespan for someone at the average age in the study population, and (c) dividing mean willingness to pay by mean expected life span. Miller (1986) identifies 25 studies of individual willingness to pay for a statistical life that are of reasonable quality. After adjusting such parameters as the value of time to make the values in the studies more comparable and adjusting for people's misperceptions of their fatality risks using the procedure in Blomquist (1982), the mean value of a statistical life across the studies was $1.95 million 1986 after-tax dollars with a standard deviation of $.5 million.

Almost all of the 25 studies involved populations with mean ages around 38. According to the Statistical Abstract (1988), the average remaining lifespan at age 38 is roughly 39 years. At a 6 percent discount rate, the value per life year at age 38 is about $120,000 or $350 per day. At a 2 percent discount rate, it is about $70,000 per year or $200 per day. By way of comparison, Moore and Viscusi (1988) estimates a statistical model of wage premiums for risk that indicates the average individual is willing to pay $90,000 for a life year and uses a 2 percent discount rate in safety decisionmaking.

The utility per year of improved health status--the second term in equation (13)--presents the greatest difficulty in valuation. Computation of differences in welfare associated with changes in health status requires knowing the utilities of alternative health states. Recent work on the measurement of health status (reviewed in the next section) provides the necessary data. This work produced scales indicating how utility loss varies with the nature and extent of functional loss.

If the utility values on a scale are normalized so that death has a value of zero and perfect health a value of one, the value associated with unit utility loss for one year will be the value of a life year. The utility in the second term is the product of the functional loss averted and the utility of this loss. To get a dollar value, this product is multiplied times the value of a functional life year.

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The third through seventh terms in equation (13) together constitute the change in human capital net of consumption that results from the health status change. This is a societal externality. The value of extra labor years and increased productivity is measured by the gain in earnings attributable to averting the illness or injury. The social costs related to health status changes essentially are medical costs borne by third-party payers, charity, or government. The seventh term is the impact of the health status change on consumption, including consumption funded by transfer payments, insurance payouts, and earnings. Under the assumption that all bequests stay within the family, the change in the family's after-tax earnings that results from the illness or injury should equal the change in the family's earnings-related consumption--so they cancel out. Thus, the externalities resulting from reduced illness or injury equal taxes gained plus transfer payments (including medical care reimbursement) averted. The dollar value of the externalities generally can be computed from the extensive literature on costs of morbid conditions and data from the Health Interview Survey.

The explicit inclusion of transfer payments in the societal benefits is consistent with the generally accepted principle that transfer payment reductions are not benefits (see, for example, Klarman, 1965 or Hu and Sandifer, 1981). Rational individuals will pay less to avoid disability if transfer payments will cover some of the associated costs. Since transfer payments were subtracted from individual willingness to pay, their explicit addition yields zero net transfers in the societal benefit estimate.

The final term in equation (13) is the value of additional children born due to the health status improvement. Arthur (1981) estimates the value of this term as -$68,125 (in 1975 dollars), based strictly on the costs society incurs per child. This approach ignores the noneconomic benefits that parents derive from their children. Analyses of direct costs and opportunity costs of children (Espenshade and Calhoun, 1986) suggest these benefits are at least as large as the opportunity costs. In this article, therefore, the net value of this term is assumed to be negligible and is ignored in the computations.

## Consistency of Empirical Estimates across Scales

The operations research and medical decision-making literature contains many scales that examine the multi-attribute utility loss associated with different health states. Some articles focus on individual diagnoses--for example, the utility loss associated with blindness or kidney failure. Others create functional ability scales and examine the utility associated with each state on the scale. Torrance (1982, 1986) evaluates the different methodological approaches used in this literature.

Tables 1 through 3 compare the utility loss that different scales suggest is associated with selected diagnoses. The studies by Green and Brown (1978), Card (1980), His et al. (1983), Miyamoto and Eraker (1985), Pliskin, Shepard, and Weinstein (1980), Sackett and Torrance (1978), and Viscusi et al. (1989) directly estimate the utility loss associated with specific diagnoses. The other loss estimates in this table were computed by developing descriptions of the functional impairments associated with the diagnoses, then computing the utility losses that each scale suggests are associated with these impairments. Impairments generally were evaluated on only a subset of the utility scales because the other scales did not include appropriate impairment categories.

This section first describes and evaluates the studies that provide utility loss estimates for at least two diagnostic conditions. Next, for each diagnosis, it compares the utility loss estimates across studies and substitutes the modal utility loss estimate into equation (13) to estimate an SCE value. This analysis is the first systematic attempt to validate the utility scales against one another or against utilities estimated from studies of specific illnesses and injuries. To provide a fairer test of the scales, we generally estimated the functional impairments on all scales first, then went back and computed the associated utility losses.

## Available Scales Showing Utility Losses

Torrance (1982) conducted a survey of 112 parents of school-age children in Canada. The survey yielded utility loss estimates for scales that evaluated four dimensions of functioning: impaired physical function, role function (ability to work, play, etc.), social-emotional function, and health function. Pain is incorporated, somewhat cursorily, in the last category. Further analysis of the original ratings and supplemental interviews yielded a multiplicative equation for combining the utility losses across dimensions of impairment (Drummond et al., 1987). The utility losses have an uncertainty range (two standard deviations) of ± 12 percent. The four impairment scales are easy to use and applied to the widest range of diagnoses of any scale we tested. The equation for combining ratings is simple and conceptually appealing; it admits the possibility of fates worse than death and recognizes that the utility loss associated with an impairment is lower if the individual initially lacked full utility because of other impairments.

Sintonen (1981) obtained ratings from 120 randomly selected Finns of the relative utility of each point on 11 functional scales: moving, hearing, speaking, seeing, working, breathing, incontinence, sleeping, eating, mental functioning, and social participation. The respondents also provided guidance on additive methods for computing a combined utility loss from the discrete losses. The method allows the analyst to go into considerable detail, which is helpful in evaluating a condition where a detailed medical description of the typical course and consequences is available. The lack of a scale related to pain detracts from rating quality, however, especially for conscious states worse than death. The large number of factors and additive weights also mean that impairments which are not systemically pervasive never are rated as very severe, which is inconsistent with the information from other utility scales.

Kind, Rosser, and Williams (1982) developed a two-dimensional scale that is particularly easy to use. One dimension measures disability, where 1 is fully mobile and 8 is unconscious. The second dimension measures distress, where 1 is none and 4 is severe. Median utility values were computed from the non-economic component of British jury awards, which follow an informal schedule. Interviews also were conducted with a non-random sample of 70

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subjects including healthy volunteers, doctors, nurses, and patients in medical and mental hospitals. The survey has methodological problems, however, in part because the 10 mental patients provided some extreme ratings that were not censored. It also is inconsistent with both other survey-based estimates of utility loss and the jury award scale. Even the jury award scale's applicability is limited because it does not deal with sensory or mental function. In addition, both the jury and survey data indicate virtually all health states involve utility losses less than 20 percent or more than 60 percent, which seems unlikely and disagrees with other studies.

Kaplan (1982) and Kaplan, Bush, and Berry (1976) provide a utility loss estimates for a scale with simultaneous dimensions of mobility, physical activity, and social activity, as well as linear score adjustments for 36 symptom-problem complexes. The scale, which was the first developed, was calibrated through a population survey in San Diego. It has the major limitation of excluding the possibility that impairments can be worse than or even almost as bad as death. In addition, the symptom-problem complexes sometimes are inconsistent; for example, why should a cough and fever add .007 to utility while a cough alone subtracts .007? Also, more analytic judgment is required to select an appropriate combination of complexes using this scale than to rate diagnoses using any of the other scales.

His et al. (1983) enlisted four physicians--specialists in orthopedics, neurology, plastic surgery, and general surgery-- then divided 476 moderate and severe injuries into their four specialty categories. The physicians defined six functional scales, with impairment levels ranging from 0 to 4: mobility, daily living (self care), cognitive/psychological, sensory, cosmetic, and pain. For each injury, the appropriate specialist rated the probable number of weeks of impairment at each level during the first year, and the probable impairment levels during the second through fifth years and thereafter. Separate ratings were done for four age groups. The impact on life expectancy and the need for corrective surgery also were estimated. Using two physicians per injury, Carsten (1986) added physician ratings of some additional injuries and redefined others, arriving at a final set of 432 injuries. Roughly 20 injury experts then used a structured computer exercise to develop weights for combining the ratings on five of the impairment dimensions (self care was omitted) into a total impairment score. Their weighting was adjusted using ratings from an American Medical Association guidebook (1984), which is discussed below. A decision by Carsten, without consulting the physicians, established that no nonfatal injury was worse than death. Luchter (1987) added the days of productivity loss as an impairment measure for minor injuries. Miller, Brinkman, and Luchter (1988) converted the workdays lost for minor injuries into utility loss estimates.

Three sources provide utility estimates for a range of diagnoses rather than for points on functional scales.

The Guides to the Evaluation of Permanent Impairment (American Medical Association, 1984) were developed by more than 100 physicians. They are intended primarily for assessing impairment through physical examination and provide guidance at a micro level. For example, (a) the impairment associated with shoulder injuries is estimated separately for the more and less dominant arms and varies with the percentage reduction in range of shoulder rotation, and (b) nine levels of impairment are presented for lung cancer. The guides also provide insight into typical impairment levels for some injuries and illnesses. The guides are imperfect. They assume nothing is worse than death. Furthermore, no central control was exerted over the influence specialists on a body system decided that system had overall functioning. Therefore, the average impairment scores for some body systems seem high.

Green and Brown (1978) asked about 100 British university students to rate the relative severity of death, selected injuries, and being unhurt in an accident. Their results are interpreted in this article as indications of the percentage utility loss during the period of disability for acute conditions and of lifetime loss for chronic and irreversible conditions.

Finally, Sackett and Torrance (1978) asked a small random sample of Canadians whether they would rather live their normal lifespan with selected chronic illnesses or live a healthy life but die prematurely. The number of years that people would trade to avoid the different impairments determined the utility losses associated with them. The conditions examined included tuberculosis, depression, renal failure, mastectomy, and an unnamed contagious disease. An important lesson of this study is that the value of an impairment rises with its permanence. More research is needed to determine (a) whether the value of avoiding minor illnesses and injuries is significantly overestimated with the approach suggested in this article and (b) how to adjust the values based on the duration of impairment.

## Estimated Investment to Reduce Selected Injuries and Illnesses

Table 1 presents estimates of the utility loss and cost associated with selected injuries. The values in the first column of data are for blindness. The utility loss estimates from Torrance (1982) and Green and Brown (1978) can be used to judge the quality of our estimates using other scales because these studies asked people about the utility loss associated with blindness; the estimates are 37 and 34 percent respectively. The 20 percent value in Card (1980) also is a survey estimate, but may not be representative of the general population because it was based on a small survey of medical personnel. We estimated a 33 percent utility loss from Carsten (1986) by doubling the estimate for losing one eye, so the estimate may be low. Our 39 percent estimate from the Kaplan (1982) scale is for someone who did not drive, walked without physical problems, was limited in choice of work, and wore glasses or had trouble seeing. These two estimates agree with the survey data. The lowest estimate, the 15 percent loss from the Kind, Rosser and Williams (1982) scale, is for a severely limited work choice but no distress. Because this description omits the sensory loss, the utility loss probably is underestimated. Sintonen (1981) provided an adjustment factor for blindness that we used in conjunction with the rating of the impact on functioning to obtain an estimated utility loss of 22 to 24 percent. This estimate may be low because blindness only affects a few aspects of functioning, which means the Sintonen scale unduly constrains the possible utility loss. Viewed from the

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perspective of the other estimates, the 85 percent utility loss estimate in the American Medical Association guide is a severe overestimate.

We conclude that the utility loss associated with blindness is probably between 33 and 39 percent. With the $1.95 million dollar value of a life, this range implies typical individuals would be willing to pay between $640,000 and $760,000 to prevent a statistical person among their group from going blind. Data on the average foregone taxes and transfer payments per blind individual should be added to this value to estimate the SCE.

The second column of data shows the utility loss associated with severe brain damage or lasting unconsciousness. Kind et al. (1982), Torrance (1984), and Green and Brown (1978) measured the utility loss associated with this injury directly and determined it was a fate 8 to 28 percent worse than death. The physician ratings in Carsten (1986) and American Medical Association (1984), which did not allow fates worse than death, rated the utility loss for unconsciousness within 5 percent of the loss for death. Sintonen (1981) found lasting unconsciousness was 3 percent worse than death. Torrance (1984) notes that the visually based rating method used by Sintonen implicitly may have indicated the survey designer expected people to consider death the worst fate, so the 103 percent utility loss may be an underestimate. Kaplan's (1982) scale does not provide good utility loss estimates for severely disabling conditions; for unconsciousness, we estimated a utility loss of 71 percent.

The studies that allow fates worse than death provide the best estimates of utility loss for lasting unconsciousness, with a 116 percent loss seeming most probable. The last three rows of data in Table 1 indicate the medical costs, lost earnings, and other public costs associated with unconsciousness (and other injuries). The medical and earnings data are from Miller, Brinkman, and Luchter (1988), while the public costs are from Miller (1986). His et al. (1983) indicates that severe head injury causes roughly a 5-year reduction in lifespan. If we use a Federal income tax rate of 23 percent (Minarik, 1985) and a state rate of 5 percent (Feenberg and Rosen, 1986), these data can be used with equation (13) to estimate the SCE for a severe head injury at $3,100,000.

As the third column of utilities in Table 1 show, complete quadriplegia is another fate worse than death, with a utility loss of 105 to 114 percent on the three reliable scales, implying a best estimate of 109 percent. The Sintonen scale did not work well here, yielding an estimated utility loss of only 49 percent because its method for combining losses does not allow a large total loss unless the sensory, mental, and motor systems all are severely affected. Kaplan's scale again worked poorly, while the physician's judged this fate almost as bad as death. Both physician judgment (Carsten, 1986) and interviews with quadriplegics who have adapted to their injuries (Torrance, 1988) indicate the utility loss may decrease over time, leveling out at about 65 percent. Complete quadriplegia reduces expected lifespan by 21.5 years according to His et al. (1983). The estimated SCE for a complete quadriplegic injury is $2,600,000.

Using the scales in Kind et al. (1982), Kaplan (1982), and Torrance (1982), we estimate the utility loss for paraplegia (data column 4) at 50 to 54 percent with incomplete paralysis and 62 to 65 percent with complete paralysis. Paraplegics surveyed by Torrance (1988) and the physicians in Carsten (1986) estimated a slightly smaller loss, around 45 percent. The students surveyed by Green and Brown (1978) and the Sintonen (1981) scale (which did not model paraplegia well) both gave estimates around 29 percent, which are probably too low. As with blindness, the utility loss in the American Medical Association (1984) guides seems much too high, 81 percent. Complete paraplegia reduces expected lifespan by 15.3 years according to His et al. (1983). The best estimate of the utility loss is 50 to 65 percent, with an SCE of $1,300,000 to $1,600,000.

For older people, severe burns (data column 5) are the worst possible fate. They typically spend the rest of their lives bedridden with sufficient pain that they cannot do simple arithmetic. Using the utility scales in Torrance (1982) and Kind et al. (1982), we estimate the utility loss at 137 to 139 percent. The physician ratings, which do not allow fates to be worse than death, yield lower and less credible values. Severe burns shorten lifespan, perhaps by about 5 years. The SCE is about $3.6 million to prevent a person in late middle age from being severely burned.

A broken lower leg (data column 6) typically causes no permanent impairment according to data from the Consumer Product Safety Commission's injury cost model (which also provided the cost data for this injury) and the physician ratings of impairment in Carsten (1986). Four of the five scales we applied suggest a broken leg will reduce utility by 30 to 36 percent in the year it occurs, while Kaplan (1982) yields an excessive estimate of 54 percent. The 34 percent estimate from Green and Brown (1978) was computed as the loss for a broken arm times the ratio of losses for amputation of a leg and an arm. With a one-year utility loss around 33 percent, the SCE for a broken leg is about $40,000.

As the last column in Table 1 shows, our ratings with the Kind et al. (1982), Torrance (1982), and Kaplan (1982) scales suggest typical minor injuries reduce utility by 36 to 38 percent for a few days. These estimates assume the number of lost work days (counting weekends as if they were workdays) equals one half of the impairment days for an employed person who is injured. The 36 to 38 percent range is consistent with survey estimates of 30 percent for a bruise and 40 percent for a sprain in Green and Brown (1978). The Sintonen scale does not work well for minor injuries, yielding a low utility loss estimate of 15 percent, because minor injuries only affect a few aspects of functioning. Including the externality costs, the SCE for a minor injury is about $1,500.

Table 2 shows estimates of the utility loss associated with selected illnesses. The first two columns of data deal with mild and severe angina. Hartunian, Smart, and Thompson (1981) provided the description of angina's impairment impacts that we used and the data on economic costs.

For mild angina, Pliskin, Shepard, and Weinstein (1980) conducted a small survey that indicated the utility loss was 12 percent, in the mid-range of the 10 to 15 percent loss estimate in the American Medical Association (1984) guides. Using the impairment scale in Kind et al. (1982), we estimated the impairment at 0.7 to 16 percent. By assuming that mild angina reduced physical and role function by half a level and also using half the pain score (severe angina caused just one level of reduction on each dimension), we estimated a 16 percent utility loss from the scale in Torrance (1982). This scale, however, did not differentiate impairment as finely as was desirable to analyze a largely asymptomatic condition. Using Kaplan's (1982) scale, we estimated an 18 percent utility loss.

For severe angina, surveys by Miyamoto and Eraker (1985) and Pliskin et al. (1980) yielded utility loss estimates of 30 to 31 percent, comparable to the estimate of 25 to 32 percent we made from the Kind et al. (1982), Torrance (1982), and Kaplan (1982) scales. The loss estimated by the American Medical Association (1984) guides is slightly higher, 35 to 40 percent.

Utility losses of 12 percent for mild angina and 30 percent for severe were used to compute SCEs of $220,000 to prevent a mild case of angina for someone age 55 and $550,000 to prevent a severe case. These estimates seem high, given the economic costs involved.

The third and fourth columns of data give estimates for food poisoning. The estimates were based on the illness descriptions and cost data in Roberts (1985). They apply to cases of salmonella and campylobacter.

Based on Roberts' description, we estimated half the severe cases involve four days of severe discomfort and inability to leave home. We estimated the other half would last six days, with three days of severe discomfort and confinement to a hospital bed and three days of severe discomfort and an inability to leave home or moderate discomfort and extreme weakness. Finally, we assumed all severe cases involve four days with no discomfort, but somewhat reduced strength and resilience. The Kind et al. (1982), Torrance (1982), and Kaplan (1982) utility scales provide consistent estimates of utility loss: 39 to 45 percent over 10 days. During the first three days, both scales indicate patients with severe cases will feel as if they would rather be dead. The SCE estimate is $2,400 to $2,600 to prevent a severe case of food poisoning.

To estimate the utility loss associated with a mild case, we made low and high estimates of impact.

- Low estimate. Assume 30 percent of the cases involve two days of severe discomfort and inability to leave home and the remaining 70 percent involve just 1.5 days of mild discomfort that is not severe enough to prevent the sufferer from going to work. Under this assumption, the average case involves a utility loss (on the Kind et al. (1982) or Kaplan (1982) scales) of 24 to 25 percent for an average of 1.65 days, with an SCE of $140 to $150. The Kaplan (1982) scale suggests an uncomfortably high 41 percent utility loss for this mild case, ascribing an overly high 33 percent utility loss to mild discomfort that does not prevent someone from working.

- High estimate. Assume 75 percent of the cases involve just 1.5 days of mild discomfort, 25 percent involve two days of severe discomfort, and 5 percent are as severe as the reportable cases. Under this assumption, the utility loss is 25 to 26 percent for an average of 2.1 days, with an SCE of roughly $200.

The SCE per day of mild food poisoning is $85 to $95. By comparison, Berger et al. (1985) obtained a mean willingness to pay to avoid a day of nausea of $91 from 18 respondents, while Gerking et al. (1986) obtained a mean of $409 from five respondents. Gerking believes that his values, and possibly even Berger's, may be higher than people actually are willing to pay. Consistent with his belief, his values exceed the values derived from the impairment scales, even though food poisoning probably is slightly worse than just feeling nauseous.

The utility loss estimates for chronic bronchitis, given in the fifth column of data, were based on a description of the course of illness developed for EPA by Viscusi et al. (1989) and were generated before Viscusi fielded his willingness-to-pay survey. Estimates we made using four scales suggest a utility loss of 35 to 45 percent. The American Medical Association (1984) guides, again high, suggest at least a 50 percent utility loss. Viscusi et al. (1989), based on a survey, estimated the utility loss at 32 percent, close to the range we predicted. Data on externality costs were not readily available to compute the SCE for chronic bronchitis.

The sixth column provides estimates of the utility loss associated with a day in the hospital. The survey by Kaplan (1982) provides a range of utility losses from 41 to 60 percent for hospitalization, depending on whether the person can move around and perform self care. Sackett and Torrance (1978) obtained an estimate of a 40 to 44 percent utility loss for hospitalization with a contagious disease. The utility loss estimates we made with the Kind et al. (1982) and Torrance (1982) scales were between 55 and 65 percent, possibly a bit high, while the 47 percent loss we estimated with the Sintonen (1981) scale was on the mark. Adding the $550 average charge for a hospital day in 1985 (from the Statistical Abstract, 1988) to a utility loss of 40 to 60 percent, the SCE per hospital day avoided is roughly $700 to $750.

The last column in Table 2 provides estimates of the utility loss associated with receiving regular dialysis for end stage renal disease. Sackett and Torrance (1978) found the loss was viewed as 60 percent by the general public and as 48 percent by those on dialysis. Again high, the American Medical Association (1984) guide estimated a 90 percent utility loss. Using the Kaplan (1982) scale, we estimated the loss at 48 percent. Using the Torrance (1982) scale, we assumed mild physical limitation; some limitation of work, with half the patients largely unable to work; frequent anxiety, but an average number of friends; a disfiguring dialysis shunt; and some discomfort. These assumptions imply a 62 percent utility loss. Without anxiety, the loss would be 50 percent. The Kind et al. (1982) scale was difficult to apply to this impairment. It suggests a utility loss of 42 to 48 percent, depending on whether the distress level is assumed to be mild or moderate. The costs associated with end stage renal disease derive from unpublished analyses by The Urban Institute, which also indicate that 10 percent of dialysis patients die each year. With a 60 percent utility loss, the SCE is $1,500,000 per case prevented.

Table 3 presents estimates of the utility loss associated with retardation, by severity. No direct survey data are available on this condition. We included it because so many public health problems, among them lead poisoning, fetal alcohol syndrome, malnutrition, foodborne listeriosis, and workplace chemical exposures, can cause children to be retarded. In the future, someone is likely to estimate willingness to pay to avoid retardation, and our estimates will be available for comparison; in the meantime, they may be useful for policy analysis.

We estimated a range of retardation levels, with a utility loss of about 20 percent associated with the need for special education, a severely limited ability to work associated with a utility loss around 50 percent, need for help in self care raising the utility loss to 55 to 60 percent, and very severe retardation raising the loss above 75 percent. The American Medical Association (1984) guides performed well in evaluating retardation, agreeing reasonably well with our ratings from the Torrance (1982) and Kaplan (1982) scales.

## A Further Comparison

The impairment estimates in the lineage from His et al. (1983) cover all possible injuries in motor vehicle crashes. Miller, Brinkman, and Luchter (1988) substitute the utility losses for fates worse than death shown here for the physician ratings, then apply the data to estimate the utility loss and associated willingness to pay to avoid a typical injury. For each diagnosis, they compute the present value of future impairment years at a 6 percent discount rate. They then estimate aggregate impairment by multiplying the impairment by diagnosis times data on 1982-1984 injury incidence derived from a sample, compiled by the National Highway Traffic Safety Administration in its National Accident Sampling System. The sample includes all injuries in roughly 30,000 crashes that were reported to the police. The aggregate impairment years next are multiplied times the $120,000 willingness to pay to save a life year. An estimated average willingness to pay to avoid injury of $12,800 results.

Insight into the quality of this $12,800 estimate, and of the impairment estimates, can be obtained from a comparison with estimates of willingness to pay to avoid nonfatal injury in the workplace. Five estimates exist that cover all reported injuries, as opposed to just lost workday injuries. All five derive from hedonic regressions that examine pay differentials for risky jobs. As Table 2 shows, four of the five estimates are between $10,500 and $13,000, satisfyingly close to the estimate from physician ratings of impairment.

The comparison between the willingness to pay to avoid motor vehicle and workplace injuries implicitly assumes that the distribution of injuries is similar in these two settings. That assumption is questionable, because back injuries occur more frequently in the workplace. A special analysis we ran of National Council on Compensation Insurance detailed claims data shows back injuries account for 30 percent of all on-the-job injuries that cause lost workdays, while Luchter (1986) indicates they account for only 5 percent of more-than-minor injuries in motor vehicle crashes. Thus, the agreement in willingness-to-pay values provides only modest confirmation of the utility loss estimates.

## Conclusion

Scales on the utility of functional impairment provide a quick, inexpensive, reasonably consistent, and theoretically supportable way to estimate SCEs for preventing a wide range of diagnoses. Using these methods requires estimating the functional impairment and reduction in lifespan associated with the health status changes. The impacts on transfer payments (including health insurance payments), administrative costs, and taxes on earnings also must be estimated.

The available utility scales yield reasonably consistent values, but these values occasionally seem unreasonably high compared to the economic costs involved (witness mild angina). Pre-planned research validating the utility losses against willingness-to-pay estimates would make it easier to use the scales with confidence.

Scales that do not allow the possibility of fates worse than death should not be used to evaluate severely disabling conditions. Torrance (1982) probably is the most reliable and flexible scale presently available, but lacks utility loss estimates for some aspects of functioning (for example, loss of reproductive capability, sustained pain) and very mild symptoms. The simplistic approach taken by Green and Brown (1978) of asking people to score relative severities of different diagnoses provided surprisingly reliable results. The American Medical Association (1982) guides to permanent impairment, which are based on physician judgment, generally overestimate utility loss.

## Table 1
## Percentage Utility Loss and Cost Associated With Selected Injuries

| Study | Blind | Severe Head | Quad | Para | Severe Burn (age 45+) | Broken Lower Leg | Minor Injury@ |
|---|---|---|---|---|---|---|---|
| Kind, Rosser, & Williams | 15 | 108 | 114 | 52-65 | 137 | 31 | 38 |
| Kaplan | 39 | 71 | 66 | 50-64 |  | 54 | 36 |
| Torrance | 37* | 116 | 105 | 54-62 | 139 | 34 | 37 |
| rehabed patients |  |  | 65* | 45* |  |  |  |
| Green & Brown | 34* | 128* | 109* | 29* |  | 30 | 30-40* |
| Card | 20* |  |  |  |  |  |  |
| Sintonen | 22-24 | 103 | 49 | 29 |  |  | 15-16 |
| Carsten | 33* | 93-100* | 85-86* | 42-45* | 91* | 36 |  |
| Am Med Assoc | 85* | 95* | 99 | 81 | 95* |  |  |
| Medical Cost | DK | 680,000 | 390,000 | 235,000 | 450,000 | 200 | 285 |
| Productivity Loss | DK | 400,000 | 210,000 | 160,000 | 100,000 | 1,350 | 280 |
| Legal, Admin, Transfer | DK | 60,000 | 60,000 | 35,000 | 60,000 | DK | DK |

@ Average daily utility loss until recovery, which occurs in less than 1 year.
* Direct measurement.

## Table 2
## Percentage Utility Loss and Cost Associated With Selected Illnesses

| Study | Angina Mild | Angina Severe | Food Poisoning@ Severe | Food Poisoning@ Mild | Chronic Bronchitis | Day in Hospital@ | ESRD |
|---|---|---|---|---|---|---|---|
| Kind et al. | .7-16 | 25-31 | 45 | 24-25 | 23-37 | 61-62 | 42-48 |
| Torrance | 16 | 32 | 39 | 25-26 | 34-45 | 55-65 | 62 |
| Kaplan | 18 | 32 | 45 | 41 | 45 | 41-60* | 48 |
| Sintonen | | | | | 30-36 | 47 | |
| Sackett & Torrance patients | | | | | | 40-44* | 60*<br>48* |
| Miyamoto & Eraker | | 30* | | | | | |
| Pliskin et al. | 12* | 31* | | | | | |
| Viscusi et al. | | | | | 32* | | |
| Am Med Assoc | 10-15* | 35-40* | | | 50+ | | 90* |
| Medical Cost | 2700 | | 60 | 1000 | DK | 500 | 250,000 |
| Productivity Loss | 50 | | 30 | 300 | DK | 50 | 90,000 |
| Transfer & Admin | 0 | | 0 | DK | DK | DK | 10,000 |

@ Average daily utility loss until recovery, which occurs in less than 1 year.
* Direct measurement.

Table 3
Utility Loss Associated with Retardation

| Condition | Util Loss | Source |
|---|---|---|
| Very severely retarded | 83 | Torrance |
| | 75+ | Am Med Assoc |
| Retarded needing help with care | 57 | Kaplan |
| | 55 | Torrance |
| | 55-75 | Am Med Assoc |
| Moderately retarded with self-care | 42-51 | Kaplan |
| | 52 | Torrance |
| | 25-50 | Am Med Assoc |
| Mildly retarded | 33 | Kaplan |
| | 20-32 | Torrance |
| | 23 | Sintonen |
| | 10-20 | Am Med Assoc |

Table 4
Willingness to Pay to Avoid Non-fatal Workplace Injuries
(1985 After-tax Dollars)

| Study | Value |
|---|---|
| Butler (1983) | $10,500 |
| Dillingham (1983) | $17,000-$26,000 |
| Olson (1981) | $12,000-$13,000 |
| Smith (1983) | $11,000 |
| Viscusi (1978) | $12,000-$21,000 |

Note: Values were converted to after-tax dollars using the method described in Miller (1986).

## NOTES

1. The societal budget constraint represents a synthesis of Lotka stable population growth dynamics with the standard Solow steady-state growth model. The equilibrium population growth rate is the solution to the integral equation of stable population theory given by:

$$1 = \int_0^{\omega} e^{-gx} p(x) m(x) dx \tag{N.1}$$

where m(x) is the female birth rate to women aged x years. The equilibrium capital-labor ratio is the solution to:

$$\dot{k} = sf(k) - gk \tag{N.2}$$

where $\dot{k}$ is rate of change in k and s is savings per worker. The comparative-static change in expected lifetime welfare ($\delta W$) resulting from a change in mortality rates across different ages ($\delta p(x)$) is found by taking the differential across equation (1):

$$\delta W = \int_0^{\omega} U[c(x),x] \delta p(x) dx + \int_0^{\omega} \partial U/\partial c(x) \cdot \delta c[\delta p] p(x) dx. \tag{N.3}$$

Under the assumptions of utility maximization and perfect capital markets the life-cycle consumption pattern is given by:

$$\partial U/\partial c(x) = \partial U/\partial c(0) e^{-gx} \tag{N.4}$$

so that

$$\delta W = \int_0^{\omega} U[c(x),x] \delta p(x) dx + \partial U/\partial c(0) \int_0^{\omega} e^{-gx} \delta c[\delta p] p(x) dx \tag{N.5}$$

The two terms in equation (N.5) can be interpreted as the change in expected lifetime welfare that come from extra years and the value of changes in the consumption pattern needed to accomodate the additional years of living. The change in consumption can be evaluated by taking differentials across the societal budget constraint, yielding:

$$0 = \int_0^{\omega} e^{-gx}c(x)\delta p(x)dx + \int_0^{\omega} e^{-gx}\delta c[\delta p]p(x)dx - (f(k)-gk)\int_0^{\omega} e^{-gx}\lambda(x)\delta p(x)dx$$

$$- \delta k[\delta p](f'-g)\int_0^{\omega} e^{-gx}\lambda(x)p(x)dx - \beta\delta g[\delta p] \qquad (N.6)$$

where

$$\beta = \int_0^{\omega} xe^{-gx}c(x)p(x)dx - (f(k)-gk)\int_0^{\omega} xe^{-gx}\lambda(x)p(x)dx - k\int_0^{\omega} e^{-gx}\lambda(x)p(x)dx.$$

$\beta$ is the life-cycle value of a marginal increase in the population growth rate (Arthur and McNicoll, 1978). Following Arthur (1981) this term can be expressed as:

$$\beta = (1/b)[\bar{c}(A_c-A_L)-kn] \qquad (N.7)$$

where b is the crude birth rate in the stable population, $\bar{c}$ is per capita consumption, $A_c$ and $A_L$ are the average ages of consumption and production, respectively, and n is the labor/population ratio. Using equation (N.6) to substitute for the second term in equation (N.5) results in the expression for the change in lifetime welfare given by equation (3) in the text.

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# Valuing Nonmarket Goods:
# A Household Production Approach[*]

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# ABSTRACT

This paper presents a unique application of the household production approach to valuing public goods and nonmarket commodities. Technical relationships are estimated between health attributes, private goods that affect health, and air quality using panel data drawn from a special survey. Statistical tests show that individuals equate marginal rates of technical substitution in household production with relevant price ratios. This result confirms theoretical implications in a particularly critical context for estimating values of health attributes and air pollution. Value estimates obtained also bear on current questions facing environmental policymakers.

## I. Introduction

Individuals frequently apply a household technology to combine public and private goods in the production of nonmarket commodities for final consumption. Hori (1975) demonstrates that in these situations, market prices of private goods together with production function parameters may encode enough information to value both public goods used as inputs and nonmarket final consumption commodities. Although this valuation methodology is objective and market based, it seldom has been applied for three reasons. First, underlying technical relations either are unknown or data needed to estimate them are unavailable. Second, even if relevant technical information is at hand, the consumer's budget surface in commodity space may not be differentiable when joint production and other complicating factors are present. As a consequence, the commodity bundle chosen is consistent with any number of marginal rates of substitution and sought after values of public goods and nonmarket commodities remain unknown. Third, joint production and nonconstant returns to scale also pose serious difficulties when taking the closely related valuation approach of estimating the area behind demand curves for private goods inputs and final consumption commodities (Pollak and Wachter 1975; Bockstael and McConnell 1983).

This paper presents a unique application of the household production approach to valuing public goods and nonmarket commodities which allows for certain types of joint production and addresses key problems identified by previous authors. Technical relationships are estimated between health attributes, private goods, and air quality. Data used in the analysis are drawn from a special survey designed to implement the household production approach.. Econometric estimates allow for truncated dependent variables in panel data using tobit models with individual-specific variance components. Key results are: (1) attempts to value detailed attributes of nonmarket home produced commodities may be ill-advised; however, estimating a common value for a broadly defined category of attributes may be possible, and (2) statistical tests show that individuals equate marginal rates of technical substitution in household production with relevant price ratios. This latter result confirms behavioral implications of the theory in a particularly critical context for estimating values of nonmarket commodities and public goods. Also, value estimates obtained bear on current questions concerning air pollution control policy. The Clean Air Act of 1970 and its subsequent amendments focus exclusively on health to justify regulation and requires air quality standards to protect even the health of those most sensitive to pollution. The survey data are sufficiently rich to allow separate value estimates for persons with normal respiratory function and persons with chronic respiratory impairments.

The remainder of this paper is divided into four sections. Section II describes a simple household production model in a health context and reviews theoretical issues in obtaining value estimates. Section III discusses the survey instrument and the data collected. Section IV presents econometric estimates of production functions for health attributes, as well as values of better air quality and improved health for both the normal and respiratory impaired subsamples. Implications and conclusions are drawn out in Section V.

## II. PRELIMINARIES

The model specifies utility (U) as a function of market goods (Z) and health attributes, called symptoms, (S).

$$U = U(Z, S) \tag{1}$$

For simplicity, Z is treated as a single composite good, but S denotes a vector measuring intensity of n health symptoms such as shortness of breath, throat irritation, sinus pain, headache, or cough. Intensity of the $i^{th}$ symptom is reduced using a vector (V) of m additional private goods that do not yield direct utility, a vector of ambient air pollution concentrations ($\alpha$), and an endowment of health capital ($\Omega$).

$$S^i = S^i(V, \alpha; \Omega) \qquad i = 1, \ldots, n \tag{2}$$

Elements of V represent goods an individual might purchase to reduce intensity of particular symptoms, and $\Omega$ represents genetic predisposition to experience symptoms or presence of chronic health conditions that cause symptoms. Notice that equation (2) allows for joint production in that some or all elements of V may (but do not necessarily) enter some or all symptom production functions.[1] The budget constraint is

$$I = P_Z Z + \Sigma_j P_j V_j \tag{3}$$

where $P_Z$ denotes the price of Z, $P_j$ denotes the price of $V_j$, and I denotes income.

Aspects of this general approach to modeling health decisions have been used in the health economics literature (e.g., Grossman 1972; Rosenzweig and Schultz 1982, 1983), where medical care is an example of V often considered. In these three papers, however, the stock of health rather than symptoms is treated as the home produced good, and Grossman treats decisionmaking intertemporally in order to analyze changes in the health stock over time. A multiperiod framework would permit a more complete description of air pollution's cumulative physiological damage, but the present model's focus on symptoms of short duration, suggests that a one period model is appropriate. Moreover, long term panel data

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containing both economic and health information necessary to assess cumulative physiological damage are difficult to obtain.

Similar models also have been used in environmental economics to derive theoretically correct methods for estimating values of air quality and other environmental attributes (e.g., Courant and Porter 1981; Harford 1984; Harrington and Portney 1987). These models, however, only consider the case in which m = n = 1 and rule out the possibility of joint production. In this situation, the marginal value of or willingness to pay (WTP) for a reduction in air pollution can be derived by setting dU = 0 and using first order conditions to obtain

$$WTP_\alpha = - U_1 S^1_\alpha/\lambda = - P_1 S^1_\alpha/S^1_1 \tag{4}$$

where $U_1$ denotes marginal disutility of the symptom, $S^1_\alpha$ denotes the marginal effect of air pollution on symptom intensity, $S^1_1$ denotes the marginal product of $V_1$ in reducing symptom intensity, and $\lambda$ denotes marginal utility of income. As shown, marginal willingness to pay to reduce symptom intensity $(- U_1/\lambda)$ equals the marginal cost of doing so $(- P_1/S^1_1)$.

Extensions to situations where m and n take on arbitrary values have been considered in the theory of multi-ware production by Frisch (1965) as well as in a public finance context by Hori (1975). Actually, Hori treats four types of household production technology. His case (3) involving joint production appears to best characterize the application discussed in Section IV because a single $V_j$ may simultaneously reduce more than one symptom. In this situation, a key result is that marginal values of symptom intensity $(- U_i/\lambda)$ cannot be re-expressed in terms of market prices $(P_j)$ and production function parameters $(S^i_j)$ unless the number of private goods is at least as great as the number of symptoms $(m \geq n)$. Intuitively, if m < n, the individual does not have a choice among some alternative combinations of symptom intensities because there are too few choice variables ($V_j$) and the budget surface on which each chosen value of $S^i$ must lie is not differentiable.[2]

Another perspective on this result can be obtained from the m first order equations for the $V_j$ shown in (5)

$$\begin{bmatrix} S_1^1 & \cdots & S_1^n \\ \cdot & & \\ \cdot & & \\ \cdot & & \\ S_m^1 & \cdots & S_m^n \end{bmatrix} \begin{bmatrix} U_1/\lambda \\ \cdot \\ \cdot \\ \cdot \\ U_n/\lambda \end{bmatrix} = \begin{bmatrix} P_1 \\ \cdot \\ \cdot \\ \cdot \\ P_m \end{bmatrix} \tag{5}$$

Each first order condition holds as an equality provided each private good is purchased in positive quantities. If m < n, the rank of the symptom technology matrix $S = \{S_j^i\}$ is at most m, the system of equations in (5) is underdetermined, intensity of one symptom cannot be varied holding others constant, and the marginal value of an individual symptom cannot be determined. On the other hand, if m = n and the symptom technology matrix is nonsingular, then the rank is n and unique solutions can be computed for the $U_i/\lambda$. If m > n and the technology matrix has full rank, then the system is overdetermined, and values for the $U_i/\lambda$ can be computed from a subset of the first order equations.

Solving (5) computes marginal values for the nonmarket commodities produced by the individual. The value of the public good input, $\alpha$, is the weighted sum of the value of the commodities, where the weights are the marginal products of $\alpha$ in reducing symptoms: $WTP_\alpha = -\Sigma_i(U_i/\lambda)S_{i\alpha}$. If the marginal products of $\alpha$ are known or estimated, solving (5) provides the information necessary to value nonmarket commodities and public goods.

This theoretical overview yields several ideas useful in empirical application. First, if $m \geq n$ and the household technology matrix has rank n, then values of nonmarket commodities and public goods are calculated in a relatively straightforward manner because utility terms can be eliminated. Second, even in cases where $m \geq n$, the household production approach may fail if there is linear dependence among the rows of the technology matrix. Thus, statistical tests of the rank of the matrix should be performed to ensure differentiability of the budget surface. Third, if $m > n$, first order conditions impose constraints on values that can be taken by the $S_j^i$; validity of these constraints can be tested. Fourth, the possibility that $m < n$ suggests that the household production approach may be incapable of estimating separate values for a comparatively large number of detailed commodities and that aggregation of commodities may be necessary to ensure $m > n$.[3]

Fifth, if $m \geq n$, values of $S_j^i$ and $P_j$ need not yield positive values for $-U_i/\lambda$, the marginal willingness to pay to reduce intensity of the i^th^ symptom. Of course, in the simple case where $m = n = 1$, the only requirement is that $-P_1/S_1^1 > 0$. If $m = n = 2$, a case considered in the empirical work presented in Section IV, values of $-U_1/\lambda$ and $-U_2/\lambda$ both will be positive only if $(S_1^1/S_2^1) \gtrless (P_1/P_2) \gtrless (S_1^2/S_2^2)$. If $V_1$ and $V_2$ are not chosen such that their marginal rates of technical substitution bracket their price ratio, then it is possible to reduce intensity of one symptom without increasing intensity of the other and without spending more on symptom reduction.

Sixth, complications arise in expressing symptom and air pollution values in situations where some or all of the $V_i$ are sources of direct utility, a form of joint production. This problem is important (and it is encountered in the empirical work presented in Section IV) because of the difficulty in identifying private goods that are purchased but do not enter the utility function. To illustrate, assume that $m = 2$, $n = 1$ and that $V_2$ but not $V_1$ is a source of both direct positive utility and symptom relief. $WTP_\alpha$ still would equal $-(P_1 S^1_\alpha / S^1_1)$ and therefore could be calculated without knowing values for marginal utility terms. If consumption of $V_2$, however, was used as a basis for this calculation, the simple formula $-(P_2 S^1_\alpha / S^1_2)$ would overestimate $WTP_\alpha$ by an amount equal to $-(U_2 S^1_\alpha / \lambda S^1_2)$ where $U_2$ denotes marginal utility of $V_2$ ($U_2 > 0$). When m and n take arbitrary values, the situation is more complex, but in general nonmarket commodity and public good values can be determined only if the number of private goods which do not enter the utility function is at least as great as the number of final commodities. Even if this condition is not met, however, it is possible in some cases to determine whether the value of nonmarket commodities and public goods is over- or underestimated.[4]

## III. DATA

Data used to implement the household production approach were obtained from a sample of 226 residents of two Los Angeles area communities. Each respondent previously had participated in a study of chronic obstructive respiratory disease (Detels et al. 1979, 1981). Key aspects of this sample are: (1) persons with physician diagnosed chronic respiratory ailments deliberately are overrepresented (76 respondents suffered from such diseases), (2) 50 additional respondents with self-reported chronic cough or chronic shortness of breath are included, (3) 151 respondents lived in Glendora, a community with high oxidant air pollution, and 75 respondents lived in Burbank, a community with oxidant pollution levels more like other urbanized areas in the U.S. but with high levels of carbon monoxide, (4) all respondents either were nonsmokers or former smokers who had not smoked in at least two years, and (5) all respondents were household heads with full-time jobs (defined as at least 1,600 hours of work annually).

Professionally trained interviewers contacted respondents several times over a 17 month period beginning in July 1985. The first contact involved administration of an extensive baseline questionnaire in the respondent's home. Subsequent interviews were conducted by telephone.[5] Including the baseline interview, the number of contacts with each respondent varied from three to six with an average number of contacts per respondent of just over five. Of the 1147 total contacts (≅ 226 x 5), 644 were with respiratory impaired subjects (i.e., those either with physician-diagnosed or self-reported chronic respiratory ailments) and 503 were with respondents having normal respiratory function.

Initial baseline interviews measured four groups of variables: (1) long term health status, (2) recently experienced health symptoms, (3) use of private goods and activities that might reduce symptom intensity, and (4) socioeconomic/demographic and work environment characteristics. Telephone follow-up interviews inquired further about health symptoms and use of particular private goods. Long term health status was measured in two ways. First, respondents indicated whether a physician ever had diagnosed asthma (ASTHMA), chronic bronchitis (BRONCH), or other chronic respiratory disease such as emphysema, tuberculosis, or lung cancer

(OTHDIS). Second, they stated whether they experience chronic shortness of breath or wheezing (SHRTWHZ) and/or regularly cough up phlegm, sputum, or mucous (FLEMCO). Respondents also indicated whether they suffer from hay fever (HAYFEV); however, this condition was not treated as indicative of a chronic respiratory impairment.

Both background and follow-up instruments also asked which, if any, of 26 health symptoms were experienced in the two days prior to the interview. Symptoms initially were aggregated into two categories defined as: (1) chest and throat symptoms and (2) all other symptoms.[6] Aggregation to two categories reduces the number of household produced final goods (n) considered; however, assigning particular symptoms to these categories admittedly is somewhat arbitrary. Yet, the classification scheme selected permits focus on a group of symptoms in which there is current policy interest. Chest and throat symptoms identified have been linked to ambient ozone exposure (see Gerking et al. 1984, for a survey of the evidence) and federal standards for this air pollutant currently are under review. Moreover, multivariate tobit turns out to be a natural estimation method and aggregating symptoms into two categories permits a reduction in computation burden. Dickie et al. (1987(a)) report that respondents with chronic respiratory impairments experienced each of the 26 individual symptoms more often than respondents with normal respiratory function. This outcome is reflected in Table 1 which tabulates frequency distributions of the total number of chest and throat and other symptoms reported by respondents in the two subsamples.[7]

In the empirical work reported in Section IV, data on the number of symptoms reported are assumed to be built up from unobserved latent variables measuring symptom intensity. As intensity of a particular symptom such as cough rises above a threshold, the individual reports having experienced it; otherwise he does not. Thus, the frequency distribution tabulated in Table 1 merely reflects the number of symptoms that crossed the intensity threshold in the two days prior to the interview.

Private goods which indicated steps taken in the past that might reduce symptoms over a period of years, measured whether the respondent has and uses: (1) central air conditioning in the home (ACCEN), (2) an air purifying system in the home, (3) air conditioning in the automobile (ACCAR), and (4) a fuel other than natural gas for cooking (NOTGASCK).[8] These variables represent goods that may provide direct sources of utility to respondents. Air conditioners, for example, not only may provide relief from minor health symptoms; but also provide cooling services that yield direct satisfaction. This problem is discussed further in Section V.

Socioeconomic/demographic variables measured whether the respondent lived in Burbank or Glendora (BURB) as well as years of age (AGE), gender, race (white or nonwhite), marital status, and household income. Also, respondents were asked whether they were exposed to toxic fumes or dust while at work (EXPWORK).

Finally, each contact with a respondent was matched to measures of ambient air pollution concentrations, humidity, and temperature for that day. Air monitoring stations used are those nearest to residences of respondents in each of the two communities. Measures were obtained of the six criteria pollutants for which national ambient air quality standards have been established: carbon monoxide (CO), nitrogen dioxide (NO2), ozone (O3), sulfur dioxide (SO2), lead and total suspended particulates. Readings for lead and particulates, however, only were available for about ten percent of the days during the study period, forcing exclusion of those pollutants from empirical work. Each of the remaining four pollutants were measured as maximum daily one-hour ambient concentrations. Maxima are used because epidemiological and medical evidence suggests that acute symptoms may be more closely related to peak than to average pollution concentrations. The air pollution variables entered then, are averages of one hour maxima on the two days prior to the interview so as to conform with the measurement of symptoms.[9] Temperature and relative humidity data similarly were averaged across two day periods.

## IV. ESTIMATES OF HOUSEHOLD SYMPTOM TECHNOLOGY

This section reports estimates of production functions for chest and throat and other symptoms. Empirical estimates of household production technology in a health context also have been obtained by Rosenzweig and Schultz (1983); however, these investigators consider determinants of birth weight rather than health symptoms and do not focus on valuing nonmarket commodities and public goods.[10] The symptom production functions reported below are estimated in a bivariate tobit framework with variance components.[11] Bivariate tobit estimation was performed because of the probable correlation between disturbances across equations. Given that symptoms often appear in clusters, individuals reporting symptoms in one category may also report them in the other. Also, as noted in the discussion of Table 1, the modal number of symptoms reported was zero.

Random disturbances follow an error components pattern, consisting of the sum of a permanent and a transitory component.

$$\varepsilon_{iht} = \mu_h + \upsilon_{iht} \qquad i = R, N \tag{6}$$

where i denotes type of symptom (chest and throat, other), h denotes respondent, and t denotes time. The transitory error component, $\upsilon_{iht}$, captures unmeasured effects that vary over individuals, symptoms, or time. The permanent error component $\mu_h$, in contrast, varies only over individuals; for a given individual it is constant over time and common to production functions for both types of symptoms. The permanent error component serves two purposes in the model. First, it captures persistent unmeasured but individual specific factors that influence symptoms, including unmeasured elements of $\Omega$ and/or the threshold at which symptoms are reported. Hence, $\mu_h$ exerts an independent influence by allowing individuals with identical measured characteristics to have different numbers of symptoms. Second, a given individual's permanent error component captures contemporaneous correlation between the two symptom classes.

The $\mu_h$ are assumed to be independent drawings from identical distributions. Mundlak (1978) and others have argued that the $\mu_h$ are likely to be correlated with values of the explanatory variables, and the error components. For example, if an individual knows his own $\mu_h$, then utility maximization would imply that his choice of private goods depends on $\mu_h$. A possible solution would be to replace the random effects with fixed effects in which the $\mu_h$ are assumed to be constants that vary across individuals. Mundlak notes, however, that the fixed effects model suffers from a serious defect if $\mu_h$ is correlated with some or all covariates: It is impossible to distinguish between the effects of time invariant covariates and the fixed effects. This defect of the fixed effects model is troublesome, because all covariates except the air pollution measures are time invariant. Since the valuation procedure of Section 2 requires distinguishing marginal products of private goods from the individual's predisposition to illness, the fixed effects model was rejected in favor of random effects.

Both transitory and permanent error components are assumed normally distributed with $E(\mu_h) = 0$, $E(\mu_h^2) = \sigma_\mu^2$, and $E(\mu_h \mu_{h'}) = 0$ for $h \neq h'$; $E(\upsilon_{iht}) = 0$, $E(\upsilon_{iht}^2) = \sigma_\nu^2$, and $E(\upsilon_{iht}\upsilon_{i'h't'}) = 0$ for $i \neq i'$ or $h \neq h'$ or $t \neq t'$. The permanent error component is distributed independently of the transitory error component, so the distribution of the summed error components is normal with $E(\varepsilon_{iht}) = 0$, $E(\varepsilon_{iht}^2) = \sigma_\mu^2 + \sigma_\nu^2$, and $E(\varepsilon_{iht}\varepsilon_{iht'}) = \sigma_\mu^2 = E(\varepsilon_{Rht}\varepsilon_{Nht})$.

Given $\mu_h$ and the distributional assumptions about the error components, the likelihood for the $h^{th}$ individual is the product of independent tobit likelihoods: one tobit for each symptom class in each time period. The conditional likelihood for the $h^{th}$ individual is

$$L_h(\mu_h) = \underset{S_{Rt}>0}{\pi} f(\upsilon_{Rt}|\mu) \underset{S_{Rt}=0}{\pi} F(\upsilon_{Rt}|\mu) \underset{S_{Nt}>0}{\pi} f(\upsilon_{Nt}|\mu) \underset{S_{Nt}=0}{\pi} F(\upsilon_{Nt}|\mu) \qquad (7)$$

where $f(\cdot)$ is the normal density and $F(\cdot)$ is the normal distribution. Conditioning was removed by integrating over $\mu$. In order to address the problem of an unequal number of interviews per respondent, log-likelihood values first were computed for each respondent, and then summed to obtain totals.[12]

Tables 2 and 3 present illustrative symptom production function estimates for the impaired and normal subsamples. Equations presented are representative of a somewhat broader range of alternative specifications that are available from the authors on request. Alternative specifications included attempts to correct for simultaneity between symptoms and private goods. Bartik (1988) calls attention to this problem in a related context and Rosenzweig and Schultz treat it in their previously cited birthweight study. Procedures devised for the present study are analogous to two-stage least squares. In the first stage, reduced form probit demand equations for each of four private goods (ACHOME, ACCAR, APHOME, NOTGASCK)[13] were estimated. In the second stage, predicted probabilities from the reduced form probits were to be used as instruments for private goods in the tobit symptom production function models. However, explanatory power of the reduced form probit equations was very poor. In half of the equations for each subsample the null hypothesis that all slope coefficients jointly are zero could not be rejected at the 5 percent level and in all equations key variables such as household income had insignificant and often wrongly signed coefficients. Another problem is the absence of private good price data specific to each respondent. The original survey materials requested these data but after pretesting, this series of questions was dropped because many respondents often made purchases jointly with a house or car and were unable to provide even an approximate answer. As a consequence, simultaneous equation estimation was not pursued further with the likely outcome that estimates of willingness to pay for nonmarket commodities and public goods may have a downward bias.[14]

In any case, one result of interest from the bivariate tobit estimates in Tables 2 and 3 is the outcome of testing the null hypothesis that estimated symptom production parameters jointly are zero. In the four equations reported, a likelihood ratio test rejects this hypothesis at significance levels less than 1 percent. Also, estimates of the individual specific error components, denoted $\sigma_\mu$, have large asymptotic t-statistics which confirms persistence of unobserved personal characteristics that affect symptoms.

Table 2 shows that chronic health ailments and hay fever are positively related to symptom occurrence among members of the impaired group. Coefficients of ASTHMA, BRONCH, SHRTWHZ, and HAYFEV are positive in equations for both chest and throat and other symptoms and have associated asymptotic t-statistics that range from 2.1 to 7.6. The coefficient of FLEMCO is positive and significantly different from zero at conventional levels in the chest and throat equation, but its asymptotic t-statistic is less than unity in the equation for other symptoms. The coefficient of AGE was not significantly different from zero in either equation and the EXPWORK variable was excluded because of convergence problems with the bivariate tobit algorithm.[15] Variables measuring gender, race, and marital status never were included in the analysis because 92 percent of the impaired respondents were male, 100 percent were white, and 90 percent were married. Residents of Burbank experience chest and throat symptoms with less frequency than do residents of Glendora. Of course, many possible factors could explain this outcome; however, Burbank has had a less severe long term ambient ozone pollution problem than Glendora. For example, in 1986 average one day hourly maximum ozone readings in Burbank and Glendora were 8.7 pphm and 10.2 pphm, respectively.

With respect to private and public inputs to the symptom production functions, the coefficient of ACCAR is negative and significantly different from zero at the 10 percent level using a one tail test in the other symptoms equation, while the coefficient of ACCEN is negative and significantly different from zero at the 5 percent level using a one tail test in both equations. Results from estimated equations not presented reveal that NOTGASCK and use of air purification at home never are significant determinants of symptoms in the impaired subsample. Also, O3, CO, and NO2 exert insignificant influences on occurrence of both types of symptoms. When four air pollution variables were entered, collinearity between them appeared to prevent the maximum likelihood algorithm from converging. Consequently, SO2 was arbitrarily excluded from the specification presented and the three air pollution measures included as covariates should be interpreted as broader indices of ambient pollutant concentrations. Variables measuring temperature and humidity were excluded from the Table 2 specification; but in equations not reported their coefficients never were significantly different from zero.

Table 3 presents corresponding symptom production estimates for the subsample with normal respiratory function. HAYFEV is the only health status variable entered because ASTHMA, BRONCH, SHRTWZ, and FLEMCO were used to define the impaired subsample. Coefficients of HAYFEV are positive in equations for both chest and throat and other symptoms and have t-statistics of 1.61 and 1.87, respectively. Coefficients of BURB are negative; but in contrast to impaired subsample results, they are not significantly different from zero at conventional levels. AGE and EXPWORK enter positively and their coefficients differ significantly from zero at 2½ percent in the other symptoms equation. Among private goods entering the production functions, coefficients of APHOME and ACHOME never were significantly different from zero at conventional levels, and these variables are excluded from the specification in Table 3. Use of air conditioning in an automobile reduced chest and throat symptom occurrences and cooking with a fuel other than natural gas (marginally) reduces other symptoms. Variables measuring gender, race, and marital status again were not considered as the normal subsample was 94 percent male, 99 percent white, and 88 percent married. In the normal subsample, collinearity and algorithm convergence problems again limited the number of air pollution variables that could be entered in the same equation. As shown in Table 3, when O3, CO, and NO2, coefficients had associated t-statistics of 1.16 or smaller. Temperature and humidity variables are excluded from the specification shown in Table 3. In alternative specifications not reported, coefficients of these variables never were significantly different from zero in alternative equations not reported.

Three pieces of information are required to use the estimates in Tables 2 and 3 in the calculation of values for nonmarket commodities (the two types of symptoms) and public goods (air pollutants): (1) marginal effects of air pollutants on symptoms, (2) marginal effects of private goods on symptoms, and (3) prices of private goods. Marginal products were defined as the effect of a small change in a good on the expected number of symptoms. Computational formulae were developed extending results for the tobit model (see McDonald and Moffit 1980) to the present context which allows for variance components error structure. However, because private goods are measured as dummy variables and, therefore, cannot be continuously varied, incremental, rather than marginal, products are used.

The final elements needed to compute value estimates are the prices of private goods. Dealers of these goods in the Burbank and Glendora areas were contacted for estimates of initial investment required to purchase the goods, average length of life, scrap value (if any), and fuel expense. After deducting the present scrap value from the initial investment, the net initial investment was amortized over the expected length of years of life. Adding annual fuel expense yields an estimate (or range of estimates) of annual user cost of the private good. The annual costs then were converted to two-day costs to match the survey data.[16] The dependent variables used in the estimated equations do not distinguish between one- and two-day occurrences of symptoms, but approximately one-half of the occurrences were reported as two day occurrences. As a consequence, the value estimates obtained were divided by 1.5 to convert to daily values.

Two tests were performed prior to estimating values of symptom and air pollution reduction. First, calculations were made for both normal and impaired subsamples to ensure that relevant ratios of incremental products of private goods in reducing symptoms bracketed the corresponding price ratio. Recall from the discussion in Section 2 that this condition guarantees that value estimates for reducing both types of symptoms are positive. A problem in making this calculation is that estimates of incremental rates of technical substitution vary across individuals (incremental products are functions of individual characteristics), but no respondent specific price information is available. As just indicated, dealers in Glendora provided the basis for a plausible range of prices to be constructed for each good. If midpoints of relevant price ranges are used together with incremental rates of technical substitution taken from Tables 2 and 3, the bracketing condition is met for all 100 respondents in the normal subsample and 117 of 126 respondents in the impaired subsample. Of course, alternative price ratios selected from this range meet the bracketing condition for different numbers of respondents.

Second, possible singularity of the symptom technology matrix was analyzed using a Wald test (see Judge et al. 1985, p. 215 for details).[17] In the context of estimates in Tables 2 and 3, the distribution of the test statistic ($\lambda$) is difficult to evaluate because relevant derivatives are functions of covariate values and specific to individual respondents. However, if derivatives are evaluated in terms of the underlying latent variable model, they can be expressed in terms of only parameters and $\lambda$ is distributed as $\chi^2$ with 1 degree of freedom. Adopting this simpler approach, p-values for the Wald test statistic are large: $p = .742$ for the impaired subsample equations and $p = .610$ for the normal subsample equations.[18] As a consequence, the null hypothesis of singularity of the symptom technology matrix is not rejected at conventional levels. This result suggests that in both subsamples, there does not appear to be an independent technology for reducing the two types of symptoms, budget constraints are nondifferentiable, and separate value estimates for chest and throat and other symptoms should not be calculated.

A common value for reducing chest and throat and other symptoms still can be obtained by aggregating the two categories and re-estimating production functions in a univariate tobit framework. Table 4 shows results based on using the same covariates as those reported in Tables 2 and 3 and retaining the variance components error structure. The Table 4 equations also make use of a constraint requiring that if $m > n = 1$, values of marginal willingness to pay to avoid a symptom must be identical no matter which private good is used as the basis for the calculation. In the case where $m = 2$ and $n = 1$, as discussed in Section II, this single value is $-U_1/\lambda = -(P_1/S_1^1) = -(P_2/S_2^1)$. In the impaired subsample, the restriction can be tested under the null hypothesis, $H_0 : \beta_{ACCAR} = (P_{ACCAR}/P_{ACHOME})\beta_{ACHOME}$, where the $\beta_i$ are coefficients of ACCAR and ACHOME in the latent model and the $P_i$ are midpoints from the estimated range of two day prices for the private goods. In corresponding notation, the null hypothesis to test in the normal subsample is, $H_0 : \beta_{ACCAR} = (P_{ACCAR}/P_{NOTGASCK})\beta_{NOTGASCK}$. Both hypotheses are tested against the alternative that coefficients of private goods are unconstrained parameters.

P-values for the parameter restrictions are comparatively large; P = .623 in the impaired subsample and P = .562 in the normal subsample. Thus, the above null hypotheses are not rejected at conventional significance levels. Respondents appear to equate marginal rates of technical substitution in production with relevant price ratios; a result that supports a critical implication of the previously presented household production model. Moreover, coefficients of private good variables defined under the null hypotheses for the two subsamples have t-statistics exceeding two in absolute value. Performance of remaining variables is roughly comparable to the bivariate tobit estimates. A notable exception, however, is that in the normal subsample univariate tobit estimates, coefficients of O3 and NO2 are positive with t-statistics exceeding 1.6. This outcome suggests that persons with normal respiratory function tend to experience more symptoms when air pollution levels are high, whereas those with impaired respiratory function experience symptoms with such regularity that there is no clear relationship to fluctuations in air quality. Intensity of particular symptoms may be greater in both subsamples when pollution levels are high, but this aspect is not directly measured.

Table 5 presents estimates of marginal willingness to pay to avoid symptoms to reduce two air pollutants. Unconditional values of relieving symptoms and reducing air pollution are calculated for each respondent from observed univariate tobit models. Table 5 reports the mean, median, and range of respondents' marginal willingness to pay to eliminate one health symptom for one day as well as mean marginal willingness to pay to reduce air pollutants by one unit for one day for the normal subsample. Symptom reduction values range from \$0.81 to \$1.90 in the impaired subsample and from \$0.49 to \$1.22 in the normal subsample with means of \$1.12 and \$0.73 in the two subsamples, respectively.[19] Also, values of willingness to pay to reduce one hour daily maximum levels of O3 and NO2 by one part per million are \$0.31 and \$0.91 in the normal subsample. Corresponding calculations are not reported for the impaired subsample because, as shown in Table 4, coefficients of air pollution variables are not significant at conventional levels.

## V. CONCLUSION

Willingness to pay values of symptom reduction and air quality improvement just presented should be viewed as illustrative approximations for two reasons. First, private goods used in computing the estimates are likely to be direct sources of utility. Second, symptom experience and private good purchase decisions are likely to be jointly determined. Nevertheless, these estimates still are of interest because aspects of joint production are taken into account. A key finding is that independent technologies for home producing symptoms are difficult to identify, thus greatly limiting the number of individual symptoms for which values can be computed. In fact, the 26 symptoms analyzed here had to be aggregated into a single group before willingness to pay values could be computed.

This outcome appears to have implications for estimating willingness to pay for nonmarket commodities in other contexts. An obvious example concerns previous estimates of willingness to pay to avoid health symptoms. Berger et al. (1987) report one day willingness to pay values for eliminating each of seven minor health symptoms, such as stuffed up sinuses, cough, headache and heavy drowsiness that range from \$27 per day to $142 per day. Green et al. (1978) present estimates of willingness to pay to avoid similarly defined symptoms ranging from $26 per day to $79 per day. In both studies, however, willingness to pay estimates were obtained symptom by symptom in a contingent valuation framework that ignores whether independent technologies are available to produce each. Thus, respondents simply may have lumped total willingness to pay for broader health concerns onto particular symptoms. Some respondents may also have inadvertently stated their willingness to pay to avoid symptoms for periods longer than one day.

Another example relates to emerging research aimed at splitting willingness to pay to reduce air pollution into health, visibility, and possibly other components. From a policy standpoint, this line of inquiry is important because the Clean Air Act and its subsequent amendments focus exclusively on health and give little weight to other reasons why people may want lower air pollution levels. Analyzing location choice within metropolitan areas, for example, may not provide enough information to decompose total willingness to pay into desired components. Instead, survey procedures must be designed in which respondents are either reminded of independent technologies that can be used to home produce air pollution related goods or else confronted with believable hypothetical situations that allow one good to vary while others are held constant.

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# ENDNOTES

1. Another, possibly troublesome, aspect of joint production occurs if some or all elements of V are arguments in the utility function. This complication is discussed momentarily.
2. Hori identifies three sources of nondifferentiability of the budget surface under joint production. The first occurs if the number of private goods is less than the number of commodities. The second arises because of nonnegativity restrictions on the private goods. This is not treated directly in the present paper, but if each private good is purchased in positive quantities, the chosen commodity bundle will not lie at the second type of kink. Hori's third cause of nondifferentiability implies linear dependence among the rows of the technology matrix.
3. Notice that this point on aggregation may apply to other valuation methods as well. Using contingent valuation surveys, for example, Green et al. (1978) and Berger et al. (1987) obtained value estimates of several specific symptoms; however, issues relating to existence of independent symptom technologies never was faced. Future contingent valuation surveys may do well to consider this point prior to eliciting estimates of willingness to pay.
4. For example, suppose $m = n = 2$ and both private goods are direct sources of utility. If equation (6) is used to solve for the $U_i/\lambda$, then: (1) if the two marginal rates of technical substitution (MRTS) do not bracket the price ratio, then the value of the commodity whose

MRTS is closer in magnitude to the price ratio will be overestimated, while the value of the other commodity will be underestimated; (2) if the two MRTS values do bracket the price ratio, then the value of either one or both of the commodities will be overestimated; and (3) in no case will the value of both commodities be underestimated.

5. Both questionnaires are presented and extensively discussed in Volume II of Dickie et al. (1987(b)).
6. Chest and throat symptoms include (1) cough, (2) throat irritation, (3) husky voice, (4) phlegm, sputum or mucous, (5) chest tightness, (6) could not take a deep breath, (7) pain on deep respiration, (8) out of breath easily, (9) breathing sounds wheezing or whistling. Other symptoms are (1) eye irritation, (2) could not see as well as usual, (3) eyes sensitive to bright light, (4) ringing in ears (5) pain in ears, (6) sinus pain, (7) nosebleed, (8) dry and painful nose, (9) runny nose, (10) fast heartbeat at rest, (11) tired easily, (12) faintness or dizziness, (13) felt spaced out or disoriented, (14) headache, (15) chills or fever, (16) nausea, and (17) swollen glands.
7. An alternative to counting the number of different symptoms experienced in the two days prior to the interview would be to consider the number of symptom/days experienced. Both approaches were used to construct empirical estimates; however, to save space, only those based on counts of different symptoms are reported. Both approaches yield virtually identical value estimates for symptom and air pollution reduction.
8. The first three private goods reduce exposure to air pollution by purifying and conditioning the air. The fourth reduces exposure because gas stoves emit nitrogen dioxide.

9. The equations also were estimated after defining the pollution variables as the largest of the one hour maxima on the two days; similar results were obtained.

10. Rosenzweig and Schultz also initially specify their production functions in translog form and then test whether restrictions to CES and Cobb-Douglas forms are justified. This type of analysis is not pursued here as most of the covariates used are 0-1 dummy variables. Squaring these variables does not alter their values. Interaction variables of course, still could be computed.

11. Although there is a linear relationship between the latent dependent variables and the private goods in the tobit model, the relationship between the observed dependent variables and the private goods has the usual properties of a production function. The expected number of symptoms is decreasing and convex (nonstrictly) in the private goods.

12. The tobit coefficients and variances of the model are estimated by maximizing the likelihood function using the method of Berndt, Hall, Hall, and Hausman (1974). The score vectors are specified analytically and the information matrix is approximated numerically using the summed outer products of the score vectors. Starting values for the coefficients and the standard deviations of the transitory error components were obtained from two independent tobit regressions with no permanent error component. In preliminary runs a starting value of unity was used for the standard deviation of the permanent error component, but the starting value was adjusted to 1.5 after the initial estimate was consistently greater than one.

13. Covariates in the reduced form regressions are: ASTHMA, BRONCH, FLEMCO, SHRTWZ, HAYFEV, BURB, AGE, EXPWORK, years of education, number of dependents, household income, and an occupation dummy variable measuring whether respondent is a blue collar worker.

14. An alternative to the two-stage procedure was suggested by Chamberlain (1980) for random effects probit models. Chamberlain's approach uses information from temporal variation in choice variables to distinguish between production function parameters and the parameters of an assumed linear correlation between choice variables and the permanent error component. The approach is not well-suited to the present study because of the lack of temporal variation in the private goods.

15. In the impaired subsample, inclusion of EXPWORK frequently caused the bivariate tobit algorithm to fail to converge. This problem arose in the specification presented in Table 2; consequently the EXPWORK variable was excluded.

16. The estimated two-day prices are: \$2.34 for ACCEN, \$1.00 for ACCAR, \$0.80 for NOTGASCK. The discount rate was assumed to be 5 percent. For further details of the procedure used to estimate prices, see Dickie et al. (1987(a)).

17. The Wald test was chosen because its test statistic can be computed using only the unconstrained estimates. Since the likelihood and constraint functions both are nonlinear, reestimating the model with the constraint imposed would be considerably more difficult than computing the Wald test statistic.

18. In other estimates of symptom production functions not reported here, corresponding p-values also are large, almost always exceeding .25 and sometimes the .80-.90 range.

19. For comparison purposes, mean values also were estimated at subsample means of all explanatory variables. Results differ little with means computed over respondents. Evaluated at subsample means, willingness to pay to eliminate one symptom for one day is $1.05 in the impaired subsample and $0.70 in the normal subsample.

## TABLE 1

## FREQUENCY DISTRIBUTIONS OF SYMPTOMS BY SUBSAMPLE

| | NUMBER OF CHEST AND THROAT SYMPTOMS EXPERIENCED IN PAST TWO DAYS | | NUMBER OF OTHER SYMPTOMS EXPERIENCED IN PAST TWO DAYS | |
|---|---|---|---|---|
| | Impaired | Normal | Impaired | Normal |
| 0 | 351 | 408 | 257 | 338 |
| 1 | 84 | 41 | 123 | 79 |
| 2 | 64 | 18 | 85 | 42 |
| 3 | 48 | 15 | 73 | 18 |
| 4 | 37 | 9 | 45 | 12 |
| 5 | 26 | 4 | 28 | 5 |
| 6 | 16 | 6 | 14 | 6 |
| 7 | 8 | 2 | 9 | 2 |
| 8 | 8 | 0 | 4 | 1 |
| 9 | 2 | 0 | 2 | 0 |
| 10 | 0 | 0 | 1 | 0 |
| 11 | 0 | 0 | 1 | 0 |
| 12 | 0 | 0 | 2 | 1 |
| 13 | 0 | 0 | 0 | 0 |
| 14 | 0 | 0 | 0 | 0 |
| 15 | 0 | 0 | 0 | 0 |
| 16 | 0 | 0 | 0 | 0 |
| 17 | 0 | 0 | 0 | 0 |
| Sample Mean | 1.348 | 0.453 | 1.668 | 0.692 |

# TABLE 2

## BIVARIATE TOBIT SYMPTOM PRODUCTION FUNCTION ESTIMATES: IMPAIRED SUBSAMPLE[a]

| | Chest and Throat Symptoms | Other Symptoms |
|---|---|---|
| CONSTANT | -3.085 | -2.043 |
| | (-3.035) | (-2.125) |
| ASTHMA | 0.8425 | 0.6724 |
| | (2.328) | (1.851) |
| BRONCH | 3.774 | 2.936 |
| | (7.663) | (6.668) |
| SHRTWHZ | 1.494 | 1.235 |
| | (3.683) | (3.428) |
| FLEMCO | 1.458 | 0.2526 |
| | (4.038) | (0.8558) |
| HAYFEV | 1.110 | 0.6613 |
| | (3.509) | (2.365) |
| BURB | -1.431 | -0.7330 |
| | (-2.728) | (-1.539) |
| AGE | 0.2986 | 2.042 |
| | (0.1596) | (1.177) |
| EXPWORK | ---[b] | ---[b] |
| ACCAR | -0.3485 | -0.4395 |
| | (-0.8885) | (-1.364) |
| ACCEN | -1.9961 | -0.6291 |
| | (-2.834) | (-1.829) |
| O3 | -0.1672 | 0.1252 |
| | (-0.5638) | (-.4475) |
| CO | 1.279 | -0.06285 |
| | (1.259) | (-0.06356) |
| NO2 | 0.5475 | 0.6384 |
| | (0.7744) | (0.9282) |
| $\sigma_\nu$ | 2.617 | 2.454 |
| | (17.70) | (20.81) |
| $\sigma_\mu$ | 1.827 | |
| | (21.17) | |
| Chi-Square[c] | 148.7 | |
| P-Value for Wald Test | 0.742 | |
| Number of Iterations[d] | 21 | |

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[a] t-statistics are in parentheses.

[b] Denotes omitted dummy variable. Also, long term health status covariates entering these equations do not represent mutually exclusive categories.

[c] The chi-square test statistic is $-2\ln\lambda$, where $\lambda$ is the likelihood ratio, for a test of the null hypothesis that the slope coefficients in both production functions are all zero.

[d] The convergence criterion is 0.5 for the gradient-weighted inverse Hessian.

## TABLE 3

## BIVARIATE TOBIT SYMPTOM PRODUCTION FUNCTION ESTIMATES: NORMAL SUBSAMPLE[a]

| | Chest and Throat Symptoms | Other Symptoms |
|---|---|---|
| CONSTANT | -5.789 | -5.479 |
| | (-2.157) | (-2.790) |
| HAYFEV | 2.316 | 1.461 |
| | (1.614) | (1.871) |
| BURB | -1.388 | -0.6248 |
| | (-1.180) | (-0.8470) |
| AGE | 4.143 | 7.075 |
| | (0.7873) | (2.091) |
| EXPWORK | 0.8707 | 1.329 |
| | (1.157) | (2.297) |
| ACCAR | -1.949 | -0.6705 |
| | (-2.905) | (-1.057) |
| NOTGASCK | -0.4613 | -0.8565 |
| | (-0.6312) | (-1.594) |
| 03 | 0.2757 | 0.3592 |
| | (0.5867) | (0.9674) |
| CO | 0.1788 | -0.07200 |
| | (0.07729) | (-0.05241) |
| NO2 | 1.841 | 1.069 |
| | (1.162) | (1.127) |
| $\sigma_\nu$ | 3.204 | 2.435 |
| | (10.15) | (11.31) |
| $\sigma_\mu$ | 1.828 | |
| | (10.44) | |
| Chi-Square[b] | 69.81 | |
| P-Value for Wald Test | 0.610 | |
| Number of Iterations[c] | 20 | |

[a] t-statistics in parentheses.

[b] The chi-square test statistic is $-2\ln\lambda$, where $\lambda$ is the likelihood ratio, for a test of the null hypothesis that the slope coefficients in both production functions are all zero.

[c] The convergence criterion is 0.5 for the gradient-weighted inverse Hessian.

## TABLE 4

## UNIVARIATE TOBIT SYMPTOM PRODUCTION FUNCTION ESTIMATES[a]

| | Impaired Subsample | Normal Subsample |
|---|---|---|
| CONSTANT | -2.253<br>(-1.263) | -6.085<br>(-2.329) |
| ASTHMA | 1.0333<br>(1.953) | |
| BRONCH | 4.649<br>(7.708) | |
| SHRTWHZ | 1.909<br>(3.242) | |
| FLEMCO | 1.769<br>(3.607) | |
| HAYFEV | 1.574<br>(3.137) | 2.216<br>(2.378) |
| BURB | -1.830<br>(-2.927) | -1.623<br>(-1.126) |
| AGE | 1.200<br>(0.4034) | 6.351<br>(1.165) |
| EXPWORK | --[b] | 1.725<br>(2.039) |
| ACCAR | -0.5900<br>(-2.585) | -1.260<br>(-2.425) |
| O3 | 0.1629<br>(0.4846) | 0.5941<br>(1.616) |
| CO | 1.013<br>(0.8041) | 0.3722<br>(0.2163) |
| NO2 | 0.8930<br>(1.130) | 1.726<br>(1.784) |
| $\sigma_\nu$ | 3.884<br>(37.29) | 3.790<br>(22.47) |
| $\sigma_\mu$ | 2.582<br>(15.84) | 2.516<br>(8.822) |
| Chi-Square[c] | 77.88 | 36.45 |
| P-Value for Parameter Restrictions | 0.623 | 0.562 |
| Number of Iterations[d] | 8 | 5 |

[a] t-statistics in parentheses.

[b] Denotes omitted dummy variable. Also, long term health status covariates entering these equations do not represent mutually exclusive categories.

[c] The chi-square test statistic is $-2\ln\lambda$, where $\lambda$ is the likelihood ratio, for a test of the null hypothesis that the slope coefficients in both production functions are all zero.

[d] The convergence criterion is 0.5 for the gradient-weighted inverse Hessian.

TABLE 5

MARGINAL WILLINGNESS TO PAY TO RELIEVE SYMPTOMS AND AVOID AIR POLLUTION

IMPAIRED SUBSAMPLE

| | Symptoms | 03 | NO2 | CO |
|---|---|---|---|---|
| Mean | $1.12 | ---[a] | ---[a] | ---[a] |
| Median | $1.09 | | | |
| Maximum | $1.90 | | | |
| Minimum | $0.81 | | | |

NORMAL SUBSAMPLE

| | Symptoms | 03 | NO2 | CO |
|---|---|---|---|---|
| Mean | $0.73 | $0.31[b] | $0.91[b] | ---[a] |
| Median | $0.70 | | | |
| Maximum | $1.22 | | | |
| Minimum | $0.49 | | | |

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[a] Denotes coefficient not significantly different from zero at 10 percent level using one tail test in estimated equations presented in Table 4.

[b] Estimates of willingness to pay for reduced air pollution do not vary across sample members. In the computational ratio, respondent specific information appears both in the numerator and denominator and therefore cancels out.

# VALUATION OF MORBIDITY REDUCTION DUE TO AIR POLLUTION ABATEMENT
## DIRECT AND INDIRECT MEASUREMENTS

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# ABSTRACT

The paper is a comparative study of alternative approaches to the valuation of a public good - air quality, in terms of its effect on morbidity levels. Three indirect approaches have been employed in the study: (1) cost of illness, (2) household health production, and (3) a market goods approach, involving the derivation of willingness to pay for clean air by exploiting the relationships among the public and market goods. The direct valuation approach encompassed several contingent valuation experiments: (1) open-ended, (2) probe biding, and (3) binary choice. The estimates of welfare change valuations derived under the various approaches are discussed and compared. The empirical analysis is based on results from a household survey, consisting of a stratified random sample of about 3,300 households from the Haifa metropolitan area (in northern Israel). It was carried out over a period of 12 months during 1986-87.

# VALUATION OF MORBIDITY REDUCTION DUE TO AIR POLLUTION ABATEMENT DIRECT AND INDIRECT MEASUREMENTS[*]

## 1. INTRODUCTION

The attributes of environmental quality, a public good, require the adoption of different valuation approaches than those customarily employed in studies of market goods. Basically, our aim is to quantify the change in consumer welfare, or benefits, measured in money units, associated with a change (an increase or a reduction) in the quantity of the environmental good (and the flow of services concurrent with this change). Willingness to pay (WTP) is the term commonly used to denote this welfare change. The monetary measures of welfare change are the compensating variation and equivalent variation, or *surplus* in the case of nonmarket goods where *quantity*, rather than price changes are involved. The compensating surplus (CS) is defined as the income change which offsets the change in utility induced by a change in the level of the public good, y, holding utility constant at its *original* level. In terms of the expenditure function, $\mu$, it is given by:

$$CV = \mu(y^0;\ P^0_X,\ V^0) - \mu(y^1;\ P^0_X,\ V^0), \qquad (y^1 > y^0) \qquad (1)$$

where the superscripts indicate initial (0), or subsequent (1), states, $P_X$ is the vector of market goods prices, V is the indirect utility function, $V(P_X, M, y)$, M is the expenditure on the market goods, and y is the public good. Analogously, the equivalent surplus (ES) is the change in income equivalent to the utility gain induced by a change in the level of the nonmarket good, holding utility at its subsequent level:

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$$EV = \mu(y^0; P_X^0, V^1) - \mu(y^1; P_X^0, V^1) \tag{2}$$

Two totally different approaches for the valuation of air quality have been used. The first employs *indirect* methods, all of which essentially attempt to infer the implicit value of the public good from observable (and presumably accurately measured) prices of private goods and services. For example, air quality affects housing prices as well as expenditures on preventive and medical care that are associated with the effect of pollution on health. Changes in air quality levels would be expected to shift the *observed* demand schedules for these market goods. From the extent and direction of these shifts, implicit prices (or marginal willingness to pay valuations) of the relevant public good might be inferred. The use of market data in the valuation of environmental goods has been expounded by Mäler (1974), Freeman (1979), and more recently and exhaustively by Bockstael, et al. (1984), and Johansson (1987). One of the indirect approaches used in this study has to the best of our knowledge seldom been used in the valuation of public goods in general, and environmental goods in particular, and in this sense constitutes a novel contribution.

"Traditional" indirect approaches employed in valuing environmental resources involved techniques such as the travel cost method (TCM), characteristically used in recreation demand studies, or the hedonic price method (HPM), which has been used to monetize urban public amenities through the analysis of housing markets (e.g., Brookshire, et al., 1982, who studied air pollution effects on property values in California). In TCM, for example, researchers have attempted to value the benefits of a public good, e.g. water quality, associated with the provision of outdoor recreation services (the latter being, at least in principle, a market good).

Household health production is another indirect method. It focuses on the consequences of health damages associated with an inadequate supply of an environmental good, such as clean air and water (e.g., Cropper, 1981, Gerking and Stanley, 1986, Berger, et al, 1987). Here one posits technical relationships between the individual consumer's health attributes, exposure to environmental pollution, and the consumption of private goods that affect health (such as medical services, or goods which help protect against exposure to health risks). The maximization of utility derived from the consumption of goods and services *and* from being healthy, given these relationships, yields an implicit value assigned by the consumer to the environmental good under study.

Closely related to the health production approach, is the "cost-of-illness" (COI) method, long used by economists and medical researchers to value the damages inflicted by environmental pollution, and hence the value attributable to improvements in the supply of environmental goods. Here one estimates the expenditure on medical services and the value of lost work and productivity associated with excess morbidity or mortality. Although easiest to apply in terms of data availability, it can be shown (Harrington and Portney, 1987) that this method yields an underestimate of the (theoretically correct) value of the public good.

Alternatively, an altogether different approach, less and less hesitantly used economists, especially in the valuation of environmental and amenity resources, is a *direct* approach, in the sense that it attempts to elicit consumers' valuations through survey interview methods. This is the contingent valuation method (CVM) - which elicits valuations within a framework of a hypothetical, contingent market for the good or service in question. The "state-of-the-art" of the contingent valuation method has been summarized by Cummings, et al (1986) and, more recently, by Mitchell and Carson (1989).

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The different approaches investigated in the present study are described in Figure 1 (the residential property hedonic model is not dealt with here, however). In this paper we apply them to the valuation of benefits derived from reducing air pollution-induced morbidity.[1] To the best of our knowledge, ours is the first comprehensive study which has employed most of the approaches currently used by economists to derive monetary values of pollution-induced health damages, based on a single, large primary micro-data base.

Figure 1

The data were collected through a household survey, carried out by the author in the city of Haifa in northern Israel, over a 12 month period in 1986-87. All the approaches employed in the study (with the exception of the residential prices hedonic model) are based on the *same* set of sample observations. This made it possible to carry out a rather comprehensive empirical analysis of the different approaches.

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Section 2 of the paper describes the study area, the survey design and the data collected, as well as presenting a number of selected epidemiological results. Section 3 deals with the CVM experimental design and valuations. Section 4 details the specific indirect market goods model employed in this study. In section 5 we present a brief description of the household health production model, and in Section 6 the results from the COI analyses, focusing on the estimation of due to production gains from reducing work losses. A comparative analysis in Section 7 sums up the alternative valuation approaches.

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[1] A survey of economic studies which have dealt with the valuation of morbidity damages associated with environmental pollution has just recently been published. See Cropper and Freeman (1988). Berger, et al. (1987) have compared CVM with COI using a small sample of Chicago and Denver residents.

## 2. DATA AND STUDY DESIGN

### 2.1 Background

Haifa, is an industrial city in northern Israel, situated on the slopes of Mt. Carmel and the adjoining Haifa Bay area. The combination of the region's topography and meteorological conditions, and a concentration of heavy industry in the lower Bay area (a power plant, oil refineries, a petrochemical complex, and others) create conditions conducive to high ambient concentrations of pollutants, especially $SO_2$ and particulates, in parts of the metropolitan region (depending on topography and wind direction) during certain periods of the year.

Maximal mean 24-hour $SO_2$ concentrations of 197 and 286 $\mu g/M^3$ were recorded in 1986 and 1987, respectively, with corresponding maximal half-hour readings of 1,271 and 2,552.[2] During the period January 1986 - April 1987, 15 violations of the absolute $SO_2$ standard were recorded in Haifa. An Intermittent Control System (ICS) which directs the area's major polluters to switch to low-sulfur fuels during environmental episodes, was activated on 23 days. In one single day, April 12, 1996, the monitoring stations registered 12 violations of the 99% standard and 2 of the 100% standard. It had been estimated that on that day alone the ICS had prevented the occurrence of at least 6 additional violation of the absolute standard! (Environmental Protection Service, 1988). It has also been noted that during the same period measurements of sulfates concentrations at certain neighborhoods (these are not taken on a regular basis) have registered a three-fold increase over those measured in 1976. High values of CO were also recorded in some areas of the city during the report period.

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[2] Currently there are two ambient standards for $SO_2$: A 99% "statistical" standard of 780 $\mu g/M^3$ (300 ppb), with a 1% exceedance level (176 half-hours per year), and an absolute standard of 1560 $\mu g/M^3$ (600 ppb). An expert committee has recently proposed converting the 99% standard into a single, 100% standard.

Concomitantly, evidence has been accumulating indicating a higher incidence and prevalence of respiratory illnesses in the area. Expansion, actual or planned, of the power and petrochemical industries has fostered the familiar conflict between economic development, regional employment and income, on one hand, and the desire for a cleaner environment, on the other hand. This, as expected, has stimulated a good deal of public controversy and media involvement.

## 2.2 The Household Survey

A household survey, based on a stratified, cluster area probability sample of about 3,600 households, in the metropolitan area of Haifa was carried out from May 1986 through April 1987. The sample was drawn from 137 Census Statistical Areas (CSA), classified into four socioeconomic groups on the basis of the latest (1983) Census. They were then further classified into three levels of ambient pollution. 16 CSAs, each approximating a different residential neighborhood, were selected to represent the 12 sampling strata. City blocks were randomly sampled within each stratum. Heads (either spouse) of all the households within each block were interviewed. The data were collected in the course of a structured interview, lasting about 30-45 minutes. The overall response rate was 81%; 9% refused to be interviewed, and another 10% could not be reached after a second visit.

Beside the usual socioeconomic and demographic data, and CVM questions (discussed below), respondents were asked about perceived air pollution levels in the neighborhood and the work place, and attitudes towards air pollution. The questionnaire included questions on self-assessed health status, present and past smoking habits of household members, and respiratory system-related symptoms and diseases of the respondent and household members. These included the following: Cough and phlegm; coughing or phlegm production first thing in the morning in summer and/or winter, and at other times of the day; and wheezing and its relationship to having a cold. Additional symptoms and diagnoses were elucidated, in relation to the respondent or other household members: Eye "infection", sinusitis, allergic irritation of nose or eyes, eczema, headache, a running nose, dyspnoea (with or without effort), pneumonia, bronchitis, and asthma (including frequency of attacks over the preceding 12-month period for the latter three). Use of medical services (primary clinic visits, medications), bed days during a two-week recall period, and hospitalization during the 12 months preceding the interview by any member of the household were also recorded.

### 2.3 Some Epidemiological Findings

A dichotomous logit model served to characterize respiratory system diseases and symptoms by fitting the model to a binary (0-1) dependent variable, where 1 indicates a reported presence, and 0 the absence of a given symptom or disease. The logit model fits the data to an equation where the dependent variable is specified as the natural logarithm of the odds, $y = \ln p/(1-p)$, $p$ being the probability of observing the phenomenon (symptom or disease) and $1-p$ the probability of not observing it, and $y$ is regressed against a set of explanatory variables. Separate equations were estimated for respondent, his or her spouse, and the family's children (the latter grouped as one observation). Thus, the fitted equation is of the form:

$$y \equiv \ln p/(1-p) = a + b \times POL + \Sigma c_i X_i . \qquad (3)$$

where POL is the variable indicating pollution level (*perceived* by the respondent, or measured) in the relevant neighborhood, and the $X_i$'s are other explanatory variables. For a dichotomous classification of neighborhood pollution (used in this analysis), an odds ratio, indicating the relative "riskiness" of a polluted neighborhood with respect to the prevalence of a given symptom or disease, is denoted by $\rho$, whose point estimate is given by

$$y(1) - y(0) = b = \ln \rho = \ln \left[ (p/1-p)_{|POL=1} \, / \, (p/1-p)_{|POL=0} \right]$$

$$\therefore \rho = e^{b} \qquad (4)$$

Thus it has been assumed that there is a constant ratio between the two odds ratios for given values of the other relevant variables, and that this ratio is *independent* of those variables when individuals with similar attributes, but residing in different neighborhoods, are compared.

Table 1 and Figure 2 give the odd ratios (in Table 1 also the upper and lower confidence intervals) for various symptoms and diseases. It should be stressed that there relationships are also controlled for smoking habits (which tend to cause similar symptoms). Only findings in which the lower 95% confidence interval is more than 1 are reported. There is a marked consistency of the findings and the significant relationship between exposure to air pollution and various measures of morbidity is clear. The analysis of data relating to the spouse of the respondent revealed similar findings. The findings in relation to the children in the households also reveal a relationship between morbidity measures and exposure to air pollution (where the smoking habits controlled for are those of the parents).

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Table 1

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Figure 2

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## 3. DIRECT VALUATIONS: CVM

### 3.1 Elicitation Technique and Analysis of Responses

Economists have long since shown that the correct measure of welfare changes due to pollution reduction, and the associated health improvements, should be based on people's willingness to pay (WTP) for pollution abatement (Schelling, 1968; Mishan, 1971). Conceptually, this measure should capture the four components which constitute morbidity damages, namely, (a) opportunity cost of time sick, (b) out-of-pocket and indirect (public) outlays for medical services, (c) defensive expenditure, and (d) psychological losses associated with suffering, pain, hedonic damages, and other direct utility losses not accounted by the first three categories. A comprehensive approach to pollution-induced health damage valuation should incorporate all four components. The money equivalent of these damages is represented by WTP for enhancing ambient air quality, through the implied reduction in exposure to morbidity risks. Of course, other benefits associated with air pollution abatement should be excluded in this case.

In the present study, pre-testing has shown that - at least in the case of Israeli respondents - questions which attempted to elicit monetary valuations for reduced morbidity (e.g., reduction in a stated number of bed days, the number of days with respiratory symptoms, or the number of acute situations during a given period), were ill received by the respondents, or they had difficulties relating to the situations described in such questions. Hence, it was imperative to state WTP in terms of reduction in pollution levels. The Israeli public in general, and in Haifa in particular, is well aware of the connection between air pollution and respiratory ailments, although of course not necessarily of the true dose-response relationships.

Interviewees were queried about the *perceived* air pollution levels in their own neighborhood. In order to provide a visual stimulus, they were shown photographs of the city of Haifa on visibly polluted and on relatively clean days.[3] They were asked to state their maximum willingness to pay for pollution abatement: (a) In order to prevent a 50% reduction of present air quality level of their neighborhood; (b) To achieve a 50% improvement in present neighborhood levels.[4] The first measure corresponds to ES (and following Randall and Stoll, 1980, will be denoted by $WTP^e$); the second corresponds to CS (denoted by $WTP^c$). The notation serves to emphasize that both are willingness to *pay* measures, not willingness to *accept* (WTA) ones. Because of the inherent difficulties in obtaining non-inflated WTA responses it was we decided against employing them in the questionnaire, given the possibility that this could have mired the WTP responses as well.[5]

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[3] The pollution levels shown in the pictures did not necessarily correspond to the indicated changes in pollution levels, and mainly served to introduce a measure of realism to the hypothetical nature of the CVM environment.

The payment vehicle was the municipal property tax, which is the sole local tax. Respondents were asked to state their WTP in terms of a percentage of the annual tax assessment (over and above their present tax assessment), by selecting the appropriate percentage figure from a payment card.[6] Respondents who were not willing to pay any sum were asked about the reasons for the zero valuation. It was thus possible to distinguish between "true" 0's, i.e. people who did not place any positive value on the improvement (or, alternatively, the prevention of deterioration), and those who implicitly registered a protest vote for a variety of reasons (objecting to the payment vehicle, believing that the polluter should pay, and so on), but who did not necessarily view

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[4] Specifically, they were instructed to refer back to the perceived level which they had previously indicated as the one prevailing in their area.

[5] On the use of WTA vs. WTP in CVM, and the controversies surrounding their derivation in empirical studies,see Bishop and Heberlein (1979); Knetsch and Sinden (1984); Gregory (1986); Mitchell and Carson (1989).

[6] Percentage categories (from 0% to 100%) were listed on the card in either ascending or descending order, vertically or horizontally These options were randomly assigned to households. The upper 100% limit did not seem to constrain the range of WTP responses. While 90% of the $WTP^e$ or $WTP^c$ values were below 100 NIS, only 0.4% of the households were in the 100 NIS or less tax bracket.

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air quality improvement as valueless[7].

The variables found to be significant in explaining the variation in $WTP^c$ and $WTP^e$ (exclusive of protest zero bids) are presented in Table 2. Since the analyses of the CVM experiments focused on the subset of positive bidders, it was necessary to correct for a possible selection bias introduced by dropping the zero responses. A procedure accounting for this bias is described in Maddala (1983).[8] The analysis proceeded in two steps. First, a probit model is used to analyze the determinants of zero bids, where the dependent variable takes a value of 1 if $WTP>0$, and 0 otherwise. In the second step positive responses are analyzed separately, with the probit model providing an estimator to correct for the selectivity effects resulting from dropping the observations with zero bids. The adjustment factor is given by the ratio $\phi(V)/\Phi(V)$, where $\phi$ and $\Phi$ are the normal probability density function and cumulative density function, respectively, and $V=b'\underline{x}$. The b's are maximum likelihood estimators from the probit analysis, and $\underline{x}$ is a vector of explanatory variables belonging to three categories: variables associated with the respondent's - or other family members' - health status, demographic and socioeconomic variables (age, sex, education, birth origin, work status, family size), and attitude shaping variables, such as perception of the authorities' involvement with pollution control, the amount of annual taxes paid, and perceived exposure to air pollution at home or at the work place. There has been an expected marked improvement in $R^2$ when the equations were estimated over the set of nonzero bid observations.

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[7] Our interpretation of the data is that although some vehicle bias exists, it has had only a limited impact upon the results. Out of about 35% of respondents whose WTP=0, 21% (for $WTP^c$) and 17% (for $WTP^e$) gave reasons which could possibly imply an objection to the payment vehicle itself ("I already pay too much tax"; "I am not willing to pay any more taxes"). Namely, altogether approximately not more than 7% of *all* respondents were affected by the vehicle to such an extent that they refused to pay any positive sum. Of course, the sums offered by other respondents may have also been affected to some unknown degree.

[8] It was applied by Kealy and Bishop (1986) in studying recreation use behavior, and by Smith and Desvousges (1987) in a CVM study on risks of exposure to hazardous wastes.

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Table 2

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The estimated regressions of nonzero $WTP^c$ and $WTP^e$ bids, for the subset of standard WTP responses (see the section below) are reported in table 2 (n=2,230). Respondents who are younger, female, and from a higher socioeconomic status tend to be willing to pay more to improve air quality, or prevent its further deterioration. Respondents who are more aware of pollution in their neighborhoods or work place, who believe too little is spent on pollution control, believe government is not too effective in controlling it, and are willing to devote of their time in public activities to this end, are also willing to contribute more towards this goal. And those who themselves, or their families, suffer from the ill health effects of pollution, are also willing to pay more to control it.

### 3.2 Contingent valuation experiments

The sampling design used in the study afforded the possibility of experimenting with alternative CVM formats, used for difference subsets of the sample, each of which could be viewed as a separate random sample from the same population. The only difference between these samples was that they were taken at different points in time. Clearly, to the extent that time of year affected the CVM responses, the statement above would have to be qualified.

The first set of questionnaires ($n \cong 2,300$), the "standard" CVM format was used, namely, an open-ended WTP question. The respondent was asked to state his or her maximum WTP for the proposed change.

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It has been suggested that a more "natural" way to conduct CVM surveys, thereby adding realism and reducing the inherent hypothetical element, is through the use of a "Buy - Not buy" choice implied by the binary choice format (Cummings, et al, 1986). To this end, a second set of questionnaires ($n \cong 450$) replaced the standard format with a binary choice format, in which respondents were asked to state whether they would be willing to pay a given percentage increase in the municipal tax for the same ±50% changes in pollution levels. The percentage categories were drawn from the pay card table, and randomly assigned to households.

To analyze these responses, behavior is usually modeled in a stochastic fashion, often by positing a random utility model to represent consumer behavior. While the binary choice format does not provide the investigator with information regarding the sample distribution of WTP valuations, it does nevertheless enable to deduce its first moments - the mean and the median. These can be compared with the corresponding statistics of the distributions obtained from the other experiments. Our analysis followed the work of Hanemann (1984) and Loehman and De (1982).

A third variant of the CVM format ($n \cong 490$) was aimed at attempting to elicit respondents' true *maximum* WTP statements, by asking them whether they would have agreed to increase - and then by how much - their initial sums had they been informed that that sum would not be sufficient to accomplish the indicated 50% change.

In the course of the survey doubts were raised whether respondents were indeed interpreting it to be a one-time payment, instead of an annual contribution, in conjunction with the payment of their annual municipal taxes. To this end, a fourth change, involving a different subset of about 400 respondents, modified the nature of the payment, from a one-time to an *annual* payment.

Tables 3 and 4 display various statistics for the four experiments, and for the overall sample: "Standard" maximum WTP, repeat bidding, binary choice, and annual vs. one-payment, for $WTP^c$ and $WTP^e$ valuations, respectively. responses. We present here the results for the analyses excluding "protest" zero-bidders (identified through the follow-up question).

Table 3

Table 4

In general, $WTP^e > WTP^c$, namely, on average respondents were willing to pay more to prevent worsening of pollution than to improve present levels. However, as noted above, unless we know the shape of the indifference curves we cannot say *a priori* whether this indeed should be the case.

Means of the binary choice format are surprisingly close to those of the standard, and especially the repeat-bid, formats. Though eliciting less information (WTP above or below a certain value, but not actual WTP itself), the resulting welfare change estimates do not very much from the standard format (particularly $WTP^e$ valuations), or from both $WTP^c$ and $WTP^e$ in the repeat bid valuations. The results suggest that, given the simplicity of the binary choice format, it should be considered first as the preferred alternative, particularly where there would not be any special interest in obtaining the sample distribution of the CVM valuations.

Regarding the repeat bidding elicitation procedure, we found a significant increase in mean $WTP^c$ and $WTP^e$, for those respondents who were willing to increase their payments (who make up only a small subset of all respondents, as one would expect), and who gave a consistent answer. We tend to interpret these results as evidence of the efficacy of this approach in deriving better WTP estimates, supporting Mitchell and Carson's (1986) advocacy of it.

We did not find significant differences between the responses of the annual and one-payment groups, supporting our suspicion that respondents processed the WTP questions in the same way they would relate to the annual municipal tax payment.

### 3.3 $WTP^c$ vs. $WTP^e$ responses

A different analysis of WTP responses is presented in Table 5, where a different grouping of mean sample values of WTP and WTP for air quality changes is presented. The table is based on responses from the subset of standard WTP questionnaires. Neighborhoods (=CSA's) were divided into the three pollution levels. With regard to $WTP^c$, it was assumed that a 50% improvement roughly implies that a neighborhood with moderate air quality would be upgraded into one with good air quality, i.e., a (relatively) clean one, and that a "bad" neighborhood would move into the "moderate" category. Similarly, with respect to $WTP^e$, a 50% deterioration in pollution levels would imply a downgrading of a relatively clean neighborhood to one with moderate levels, and so on.[9] Thus, on average, an individual living in a moderately polluted neighborhood (according to his or her perception) would be willing to contribute NIS 37.9 annually towards improving air quality, and NIS 40.0 in order to prevent a worsening of present levels.

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Table 5

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The relationship between these two welfare change measures for any given sub-sample of neighborhood households is ambiguous. While $WTP^e > WTP^c$ for moderately polluted neighborhoods, the reverse holds for those badly polluted. However, both $WTP^c$ and $WTP^e$ increase with pollution levels, and the between-group differences are significant (non-parametric median test). The two-sample mean tests indicate that although $WTP^c$ and $WTP^e$ differ significantly, $WTP^c > WTP^e$ in one case (respondents from poor-quality neighborhoods), but the reverse holds for moderate-quality neighborhoods.

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[9] The neighborhood marked "Very poor" in Table 5 is a fictitious neighborhood, created by hypothetically downgrading the "poor" neighborhood category.

### 3.4 Reliability of CVM valuations

Doubts about the truthful revelation of preferences obtained through direct questioning procedures still dominate many discussions involving the use of direct WTP valuations. Four "Reference Operating Conditions" (ROC's) have been proposed by Cummings, et al (1986), as criteria for evaluating CVM applications in general, and for evaluating the accuracy of the values obtained in particular. These conditions are (a) familiarity with the commodity, (b) prior valuation and choice experience with respect to consumption levels of the commodity, (c) the presence of little uncertainty and, (d) the use of WTP, rather than WTA (willingness to accept) valuations.

In examining these conditions in the context of the present study, we note first that the city of Haifa and its environs provide a suitable setting for obtaining WTP responses in a CVM environment. Its topographical layout and the location of its industry introduce inter-neighborhood variability in ambient air quality, about which there is a fair level of public awareness. In recent years, the local media have frequently addressed the issue of air pollution-induced diseases. It is therefore likely that respondents were not placed in a position of having to respond to hypothetical CVM questions. Moreover, it has been surmised that a willingness to pay for air pollution abatement would tend to involve little or no strategic biases attributed to CVM surveys, because relatively small sums of money (per household) are typically involved. Thus, of the four conditions noted above, the first and the last have been satisfied in this study. [10]

Regarding ROC #2, all that can be claimed is that subjects were familiar with the vehicle (city property tax assessments), although, naturally, they had had no prior experience with valuing air quality in this particular manner. However, it is doubtful whether ROC #3 was fulfilled in this study. First, uncertainty is ingrained in dose-response relationships between air pollution and health, especially when lay people are involved. Secondly, an altogether different type of uncertainty may have surrounded the stipulated change in the supply of the "paid-for" commodity (the indicated level of air quality improvement), had the payment indeed been made. Although the phrasing of the relevant question attempted to alleviate this source of uncertainty, we have no way of ascertaining whether this had been successfully achieved.

## 3.5 Population CVM Estimates

Population estimates of $WTP^c$ and $WTP^e$ for the *entire* Haifa metropolitan region, were derived using the following entities:

$N_{is}$ = The number of households in the i-th CSA by employment status (s) of the head of the household (employed, self-employed, and unemployed).

$I_s$ = Average *net* monthly income per household of households whose heads were employed (Central Bureau of Statistics, 1985b). Since income of self-employed by CSA is not available, it was determined on the basis of sample means, after proper adjustments. Income levels were converted to 1987 NIS using the Cost-of-Living Index and the change in real income of salaried workers (Bank of Israel, 1988).

All census areas were classified by socioeconomic level (e)

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[10] Indeed, the survey indicates that subjects were highly familiar with the various pollution levels in their respective neighborhoods. As noted in an earlier footnote, a high partial correlation between measured and perceived pollution levels is evident.

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and pollution level (p), corresponding to those used in delineating the sampling strata. Using these data, $WTP^c$ and $WTP^e$ totals for each CSA, were derived by grouping all CSA's (sample and non-sample) according to their respective socioeconomic level (e) and pollution level (p). Each CSA was further sub-divided by employment status. The corresponding sample CSA mean WTP value was used for calculating population totals for each sub-group within each CSA. Regional totals were then obtained by aggregating employment-group totals within each CSA, and then aggregating over all CSA's . Total regional annual benefits of pollution reduction ($\Sigma WTP^c$) and of prevention ($\Sigma WTP^e$) amounted to NIS 3.9 and 9.9 mil., respectively (at the then prevailing exchange rate of 1.5 NIS to $ 1 US, $2.6 and 6.6 mil.)

## 4. INDIRECT VALUATION: DERIVING EXACT WELFARE CHANGE MEASURES

### 4.1 Introduction

In calculating benefits associated with a larger supply of the environmental public good through its relationship with some market good(s), one might begin with estimating a demand function for the market good from observed price-quantity data. The benefits from the public good would be derived by computing the change in consumers' surplus associated with a corresponding shift in the *market* demand schedule. This method would be expected to yield an *approximate* value of the potential welfare change (Just, et al, 1982). Alternatively, *exact* (in the theoretical, not statistical, sense) measures of welfare change may be obtained by evaluating an expenditure function underlying the ordinary market-good demand system, using duality theory (Hausman, 1981; Vartia, 1983; Loehman, 1986). This approach is discussed in this section.

In order to eventually "untangle" the demand valuations of the public good from those observed for the market goods, the posited demand system ought to satisfy two conditions. The market and nonmarket goods must be non-separable, and a price vector which would drive the marginal utility from the nonmarket goods to

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zero should exist (Mäler, 1974). These conditions enable the recovery of the preference ordering for this group of goods and, subsequently, the compensated demand (or marginal willingness to pay) schedule for the public good, from which valuations of changes in the quantity of that good can be derived. The demand system specified below satisfies the first condition; the second condition is not testable, but assumed.

Specifically, in this study a twice differentiable indirect utility function was assumed. Duality theory (Roy's identity) is invoked in deriving the corresponding budget share equations. This *partial* system[11] encompasses two market goods, housing services and medical services, denoted by the vector X in the formulation below, and a public good, air quality, denoted by y. The expenditure function, derived from the posited indirect utility function, is then used to calculate the monetary value of welfare changes associated with shifts in the level of air quality. By Shephard's Lemma, the partial derivative of the expenditure function with respect to price yields a Hicksian compensated demand function (cf. Varian, 1984); the derivative with respect to the public good yields the demand "price" function for the public good.

We know of only one recent study which adopted a similar, indirect market good approach to the *empirical* estimation of the benefits associated with an environmental good (Shapiro and Smith, 1981). Our paper differs in its use of individual, micro data, as compared to their use of aggregate data, and in deriving exact welfare measures (which was not the focus of that paper). In connection with measuring cost of living changes, Cobb (1987) has used a "translating variables" specification in incorporating nonmarket goods in budget share equation systems.

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[11] Partial demand system are frequently encountered in empirical studies. This is characteristically due to data limitations which preclude the estimation of all the unknown parameters in the complete demand system. In order to recover the preferences for the nonmarket good from the *partial* system it is necessary to assume that the group of commodities which make up the partial system is separable in consumption from all other commodities (Hanemann and Morey, 1987). These authors go on to show that the compensating and equivalent measures calculated from a partial demand system need not be identical with those calculated from a full system. CV would be a lower bound on the conventional compensating measure, while EV might be greater than, less than, or equal to the full system measure.

### 4.2 Model specification and estimation

The specification chosen for the indirect utility function is the translog function (Christensen et.al., 1975), defined in terms of normalized prices of the two market goods, $P^{*}=P_1/M$, the nonmarket good - air quality - y, and household characteristics:

$$
\begin{aligned}
\ln V = {} & \alpha_0 + (1 + \ln y) + (\alpha_1 + \gamma_1 \ln y) \ln P_1^{*} + (\alpha_2 + \gamma_2 \ln y) \ln P_2^{*} + \\
& + \frac{1}{2}\Big[(\beta_{11} + \delta_{11} \ln y)[\ln P_1^{*}]^2 + (\beta_{12} + \delta_{12} \ln y)\ln P_1^{*} \ln P_2^{*} \\
& + (\beta_{21} + \delta_{21} \ln y)\ln P_1^{*} \ln P_2^{*} + (\beta_{22} + \delta_{22} \ln y)[\ln P_2^{*}]^2\Big] \\
& + \ln P_1^{*} [\phi_{11}h_1 + \phi_{12}h_2 + \phi_{13}h_3 + \phi_{14}h_4 + \phi_{15}h_5] \\
& + \ln P_2^{*} [\phi_{21}h_1 + \phi_{22}h_2 + \phi_{23}h_3 + \phi_{24}h_4 + \phi_{25}h_5] + \Sigma\, \phi_i h_i
\end{aligned}
\tag{5}
$$

where $P_1^{*}$ is the (normalized) price of housing services, and $P_2^{*}$ is the (normalized) price of medical services. The $h_i$'s are dichotomous variables which represent family or head of household health characteristics: $h_1$ - smoking habits, $h_2$ - respiratory illness symptoms (head of household), $h_3$ - respiratory illness symptoms (all other household members), $h_4$ - respiratory diseases (head of household), and $h_5$ - respiratory diseases (all other household members).

By Applying Roy's identity to eq. (5) the following share equations are derived:

$$
- \frac{\partial \ell n V}{\partial \ell n P_i} \Big/ \frac{\partial \ell n V}{\partial \ell n M} = S_i \equiv \frac{PX_i}{M} \quad =
$$

$$= \left\{(\alpha_i+\gamma_i \ln y) + (\beta_{ii}+\delta_{ii}\ln y)\ln P_i^* + \frac{1}{2}(\beta_{ij}+\delta_{ij}\ln y)\ln P_j^* + \frac{1}{2}(\beta_{ji}+\delta_{ji}\ln y)\ln P_j^* + \sum_{k=1}^{5} \phi_{ik}h_k \right\} \Big/ D \qquad i=1,2 \qquad (6)$$

where

$$D = (\alpha_i+\gamma_i \ln y)+(\alpha_j+\gamma_j \ln y) + (\beta_{ii}+\delta_{ii}\ln y)\ln P_i^* + (\beta_{jj}+\delta_{jj}\ln y)\ln P_j^* + \frac{1}{2}(\beta_{ij}+\delta_{ij}\ln y)(\ln P_i^* + \ln P_j^*) + \frac{1}{2}(\beta_{ji}+\delta_{ji}\ln y)(\ln P_i^* + \ln P_j^*).$$

$S_i$ is the share of the ith market good in total expenditures, M. Symmetry constraints (analogous to the integrability condition from demand theory, see Christensen, et al., 1975) have been imposed on the demand system, *viz.*, $\beta_{ij}=\beta_{ji}$, $\delta_{ij}=\delta_{ji}$ and $\phi_{1k}=-\phi_{2k}$ for all k, causing the characteristic variables to drop out of D above (*cf.* Jorgenson and Slesnick, 1987).[12] Furthermore, the budget share equations should be homogeneous of degree zero in the parameters. To this end, a convenient normalization which guarantees this condition is $\sum_i \alpha_i=-1$ (*cf.* Christensen and Manser, 1977). The demand system should also satisfy the adding-up restriction, $\sum_i S_i=1$, which implies that the parameters of the second equation in our two-equation system, can be determined from

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[12] Note that after some rewriting, equation (6) takes the following form:

$$S_1 = \{\ldots + [\tfrac{1}{2}(\beta_{12}+\beta_{21}) + \tfrac{1}{2}(\delta_{12}+\delta_{21})\ln y]\ln P_2^* + \ldots\}/D$$

$$= \{\ldots + (\beta_{12}^*+\delta_{12}^*\ln y)\ln P_2^* + \ldots\}/D$$

$$S_2 = \{\ldots + (\beta_{21}^*+\delta_{21}^*\ln y)\ln P_1^* + \ldots\}/D$$

where $\beta_{12}^*=1/2(\beta_{12}+\beta_{21})$, etc. Since the parameters $\beta_{12}$, $\beta_{21}$, $\delta_{12}$, and $\delta_{21}$ are not identifiable when estimating the share equations, the parameters $\beta_{12}^*=\beta_{21}^*$ and $\delta_{12}^*=\delta_{21}^*$ are estimated instead. To simplify notation, however, the asterisks have been *suppressed* in the rest of the paper.

those of the first system; hence, only one equation needs to be estimated. We may note that the present data base has made it possible to incorporate individual health characteristics, related to respiratory illnesses and symptoms, into the posited preference function.[13] In this sense, the present indirect valuation can also be likened to the household health production approach used to evaluate morbidity and mortality benefits (see below).

Annual municipal tax assessments were used as proxies for housing prices in the estimation of the budget share (eq. 6). Its rates generally reflect dwelling quality and the socioeconomic status of the neighborhood. This variable was used instead of imputed rental value because there are no reliable, published statistics on housing prices by neighborhood and housing quality. Consumption of housing services has been assumed to be given by dwelling size.

The price of medical services was calculated as a weighted index of national, average estimates of primary clinic cost per patient visit and hospitalization costs for all illnesses. Consumption of medical visits was given by a predicted number of clinic visits, derived from a logit regression analysis of the survey data.[14] Hospitalization data were taken directly from the questionnaire, where respondents were asked to indicate whether they had been hospitalized for respiratory system-related illnesses during the 12-month period preceding the interview.[15] The $h_k$'s are health attributes of the respondent (head of household) or other household members, that are presumed to be associated with, or induced by, air pollution (with the exception of smoking which itself induces similar symptoms). The health variables include coughing, wheezing, sputum emission and shortness of breath; diseases refer to asthma, bronchitis, pneumonia, and other lower respiratory tract diseases. As already indicated, y stands for the perceived level of neighborhood pollution. Respondents were requested to indicate this on a severity scale of 1 to 6.[16]

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[13] For the inclusion of characteristics in an indirect translog utility function, see Woodbury (1983), in connection with a model describing labor compensation. The characteristics there are parameters which describe the worker or the work place. In a similar vain, Morey (1985) incorporated personal and site attributes in estimating a demand system for ski resorts (see also Jorgenson and Slesnick, 1987).

[14] Respondents were asked whether they visited a clinic during a two week recall period prior to the date of the interview. The logit regressions yielded predicted probabilities of at least one visit during the two week period as a function of socioeconomic and health characteristics, and a seasonal variable. These probabilities were then converted into an expected annual number of visits for each household.

To estimate the share equation (6) we employed a procedure that combines iterative minimization methods for non-linear regression with OLS estimation, imposing the symmetry and adding-up restrictions. All variables were normalized through division by their respective sample mean. Table 6 displays the parameter estimates. Inserting the parameter estimates from the budget share (5) into the indirect utility function (4), and evaluating its partial derivatives with respect to prices, income, and the public good, at the point of means, it can be shown that $\partial V/\partial P_i^* < 0$ (i=1,2), $\partial V/\partial M > 0$, and $\partial V/\partial y > 0$, as expected. Utility decreases with a rise in the (normalized) prices of housing and medical services, and rises with the level of money expenditure on the two market goods and with the level of air quality. It can also be shown that the function possesses the correct signs for the second derivatives.

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[15] It should be noted that the majority of families belong to one of several quasi-public health insurance schemes, and do not pay directly for medical services. However, paying for private medical visits and medications in order to obtain faster, and often better quality treatment is quite common, especially with sick children. Information on these extra costs, available from the survey, was also used in deriving expenditure levels. It can therefore be surmised that the number of clinic visits, in and by itself, reflects an opportunity cost of time in obtaining medical treatment, even though no immediate payment is necessarily associated with it.

[16] While the perceived level of pollution may directly affect the demand for housing and hence values, its impact upon medical expenditure is indirect; the latter are affected by actual pollution levels. However, there is a rather high partial correlation between these two measures (r=0.77). On the appropriateness of using perceived rather than actual measures of pollution levels from a psychological perspective, see Zeidner and Shechter (1988). It may be noted that had it been possible to elicit quantitative responses for perceived air quality, it probably would have been possible to use the restricted indirect utility function as suggested by Diewert (1978).

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Table 6

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### 4.3 Welfare change measures

The expenditure function takes on the form:

$$\mu = \Bigg\{ - \Big[a_1 + a_2 + (\ln P_1)(b_1 + b_2) + (\ln P_2)(b_2 + b_3)\Big]$$

$$\pm \Bigg\{\Big[\Big[a_1 + a_2 + (\ln P_1)(b_1 + b_2) + (\ln P_2)(b_2 + b_3)\Big]^2$$

$$- (2b_1 + 4b_2 + 2b_3)\Big[\alpha + (\ln P_1)(a_1 + d_1) + (\ln P_2)(a_2 + d_2) + \frac{1}{2} b_1 (\ln P_1)^2 +$$

$$+ b_2 \ln P_1 \ln P_2 + \frac{1}{2} b_3 (\ln P_2)^2 - \ln V\Big]\Big]\Bigg\}^{\frac{1}{2}}\Bigg\} \left[\frac{1}{b_1 + 2b_2 + b_3}\right] \qquad (7)$$

where $\mu = \ln M$, $\alpha = \alpha_0 + 1 + \ln y$, $a_1 = \alpha_1 + \gamma_1 \ln y$, $a_2 = \alpha_2 + \gamma_2 \ln y$, $b_1 = \beta_{11} + \delta_{11} \ln y$, $b_2 = \beta_{12} + \delta_{12} \ln y$, $b_3 = \beta_{22} + \delta_{22} \ln y$, $d_1 = \sum_{k=1}^{5} \phi_{1k} h_k$, and $d_2 = \sum_{k=1}^{5} \phi_{2k} h_k$.

Given the parameter estimates from eq. (6), CS and ES values (eqs. 1 and 2) - associated with a ±50% shift from the baseline air quality levels - can be calculated using eq. (7). These calculations yielded annual payments of 2.33 and 105.10 NIS, respectively, per household. Because the expenditure function is nonlinear, the values which have just been calculated are equivalent to evaluating a function of the form $f(\bar{x})$, which generally would not yield the same values obtained from evaluating $\overline{f(x)}$ instead. Thus, we have also computed the means of *individual* valuations by calculating the two welfare measures for each household, using the relevant attributes for that household. These calculations yielded the following mean valuations for the sample of households: $WTP^c = 9.81$ NIS (s = 38.3), and $WTP^e = 73.25$ (s = 106.2). As noted above, and shown by Loehman (1986), there is no a *priori* theoretical justification for expecting either EV>CV or the reverse; both cases are consistent with theory, and the direction of the inequality sign depends on the shape of the indifference curves.

The expenditure function for utility kept at a level associated with the initial (sample mean) air quality is shown in Figure 3 (on a logarithmic scale). The corresponding Bradford-type bid curves, showing WTP as a function of y for utility held at the initial level (CS), and at the final level (ES), are drawn in Figure 4 (marked $WTP^c$ and $WTP^e$, respectively). It can be seen from Figure 3 that the marginal bid function, or the compensated demand for the public good (the partial derivative of the expenditure function with respect to the public good, for given market good prices and utility level), would be negatively sloped.

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Figure 3
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Figure 4
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## 5. INDIRECT VALUATION: HEALTH PRODUCTION APPROACH

### 5.1 Introduction

The household health production is the basis of a valuation approach in which the benefits from a public good, viz., environmental quality, are assessed indirectly through household optimizing behavior with respect to the production (and consumption) of good health. This health "capital" is an argument in the utility function, along with other goods and services. The production of health contributes to utility on two counts: (1) Reducing expenditures on health care services, which otherwise would have decreased the amount of income available for spending

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on utility-enhancing goods and services[17]; (2) Diminishing the impact on utility through income reduction caused by work-loss days, or increasing income through productivity gains. In this framework,one would also have to consider decisions concerning the money time spent on preventive or averting activities. These contribute directly to the production of health stock (but also reduce the budget available for goods and services). Of course. the total effect on utility amounts to a WTP valuation of the welfare changes attributable to changes in the quantity of the environmental good.

Several studies have used the health production approach to estimate the value of reducing health risk resulting from air pollution abatement (e.g., Cropper, 1981; Gerking and Stanley, 1986; Harrington and Portney, 1987; Berger, et al., 1987; Dickie and Gerking, 1988). The emphasis has been on the inclusion of preventive expenditure in a utility maximizing framework, and demonstrating the theoretical superiority of this approach compared to the COI approach. The latter overlooks preventive expenditure, namely, the possibility that individuals yield a measure of control over the state of their health, any direct utility losses associated with illness, and the value of bed-day losses of the non-working population (cf., e.g., Cooper and Rice, 1976). It should be noted, however, that in the various empirical applications of the health production approach, the budget-reducing or income-enhancing effects have generally been not explicitly considered, and a fixed budget is assumed. What one is left with is usually a utility maximizing framework where only preventive activities (in addition to medical care and other consumption expenditure) are taken into account (see the empirical sections of the above cited studies).

In this section we outline a model which attempts to provide a comprehensive framework for dealing with uncertainty[18] and the dynamic aspects of the health production process. Since we too assume a fixed budget, our approach yields valuations of the environmental good which do not take into consideration the labor savings component. We only outline the model here (for a full description see Shechter, 1988), and then provide some tentative WTP estimates.

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[17] To the extent that the utility derived from consumption of goods and services is in turn affected by health conditions, then reduction of bed days would also be taken into account.

## 5.2 The model

Assume an individual producing different levels of health depending upon initial health stock, the amount of medical or preventive care consumed, the level of the environmental public good, and socioeconomic attributes. Uncertainty is represented by probabilities of being in an ill or a healthy state, following a first-order Markovian process (Hey and Patel, 1983). Several simplifying assumptions, some quite strong, have been made: (1) The probabilities are a function of the individual's current health state and not affected by age or by past medical history. (2) Two types of health stock related expenditure exist: Preventive care and medical care, where the former is exercised only when the individual is healthy, while the latter is consumed only when he or she is ill.

The health production process is given by:

$$H = \begin{cases} H_h(m_h,\ y,\ \sigma) & \text{when healthy} \\ H_s(m_s,\ y,\ \sigma) & \text{when ill} \end{cases} \qquad (8)$$

$$\frac{\partial H_h}{\partial m_h} > 0\ ;\ \frac{\partial H_s}{\partial m_s} > 0\ ;\ \frac{\partial H}{\partial y} > 0 \text{ and } \frac{\partial H}{\partial \sigma} \lessgtr 0 \text{ (for h and s)};\ \frac{\partial^2 H_h}{\partial m_h^2} < 0;\ \frac{\partial^2 H_s}{\partial m_s^2} < 0$$

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[18] A different approach to uncertainty is given by Berger, et al. (1987).

where

H - the individual's health level,

$m_h$ - amount of preventive care consumed,

$m_s$ - amount of medical care consumed,

y - the level of the environmental good,

$\sigma$ - socioeconomic characteristics of the individual.

The budget constraint is:

$$I = \begin{cases} XC_x + m_h \cdot C_h & \text{when healthy} \\ XC_x + m_s \cdot C_s & \text{when ill} \end{cases} \tag{9}$$

where

I - income

X - a composite good which does not affect health,

$C_x$ - the price of X (normalized to $C_x=1$),

$C_h$ - the price of a unit of preventive care,

$C_s$ - the unit price of medical care.

A state-dependent utility function is defined over two goods - the composite good, x, and health , h.

$$U(x;\ h)= \begin{cases} V(x) & \text{when healthy} \\ W(x) & \text{when ill} \end{cases} \tag{10}$$

It is assumed that for any given x, utility in the healthy state is greater than in the illness state, $V(\cdot)>W(\cdot)$, and that the individual is risk averse: $V'>0$, $V''<0$, $W'>0$, and $W''<0$.

For the Markovian process of transition between health states

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over time the following probabilities have been defined:

P - The probability that an individual who is healthy today will also be healthy in the next period, where $P'(h) > 0$;

1-P - The probability that an individual who is healthy today will be ill in the next period;

Q - The probability that an individual who is ill today will be healthy in the next period, $Q'(h) > 0$;

1-Q - The probability that an individual who is ill today will also be ill in the next period.

## 5.3 Optimization

The individual is assumed to maximizes lifetime expected utility, allocating the budget among X, $m_h$ and $m_s$, given the health production function and the budget constraint. Expected lifetime utility from T onward is given by

$$\sum_{t=T}^{\infty} \rho^{t-T} U_t(X;H) \tag{11}$$

where $\rho$ is the rate of time preference.

One first solves for the optimal values of X, $m_h$ and $m_s$, subject to the constrains. As noted, these optimal values are time-invariant, implying that all time periods are identical, given the state of the individual' health. Upon totally differentiating the first order condition, it is possible to obtain an expression for the individual's willingness to pay for a change in the level of the environmental good, $\frac{dI}{dy}$, measuring the value at the margin of the public good after all utility-maximizing, consumption adjustments have been made. We omit the details of the derivation (see Shechter, 1988), and give the final expression:

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$$\frac{dI}{dy} = \frac{- P'\frac{\partial H_h}{\partial y} - Q'\frac{\partial H_s}{\partial y}}{\frac{P'}{C_h}\frac{\partial H_h}{\partial m_h} - \frac{Q'}{C_s}\frac{\partial H_s}{\partial m_s}} \tag{12}$$

Note that expressions involving utility terms have been factored out, facilitating in principle empirical applications (cf. Gerkings and Stanley, 1986; Berger, et al., 1987).

We would generally expect $\frac{dI}{dy}$ to be negative, because a decrease in air quality would require some compensation for utility (at the optimal level) to remain unchanged. The change would increase health risks and welfare losses, even after the individual makes an attempt to offset this increase, at least partially (depending on one's preferences), through some budget reallocations entailing, among others, more spending on preventive or medical care. For $\frac{dI}{dy}<0$, the following conditions, - which seem reasonable - should simultaneously be satisfied:

(a) $W'>V'$ -- the marginal utility of income of a non-healthy individual is higher than that of a healthy individual.

(b) $P'\frac{\partial H_h}{\partial y} < Q'\frac{\partial H_s}{\partial y}$

That is, the change in the probability of being healthy in the next period due to a change in air quality is higher for an ill person than for a healthy one.

In order to apply the model to available data, an additional simplifying assumption was made, namely that there is no distinction between medical and preventive activities, and both having the same unit price. Thus:

$$m_h = m_s, \quad \left(\frac{\partial H}{\partial m_s} = \frac{\partial H}{\partial m_h} = \frac{\partial H}{\partial m}\right), \quad C_h = C_s = C, \quad \frac{\partial H_h}{\partial y} = \frac{\partial H_s}{\partial y}.$$

From this it follow that

$$\frac{dI}{dy} = - \frac{\left[\frac{\partial H}{\partial y}\right] (P'-Q')}{\frac{1}{C}\frac{\partial H}{\partial m} \quad (P'-Q')} = - \left[ \frac{\frac{\partial H}{\partial y}}{\frac{\partial H}{\partial m}} C \right] \tag{13}$$

The simplifying assumptions eliminate the rationale for the transition probabilities, since no distinction is effectively made between two states of health. It is immediately seen that these simplifying assumptions render the results identical with those obtained by Gerkings and Stanley (1986) and Berger, et al. (1987). It is also readily seen that the amount of money an individual is willing to substitute for a given improvement of air quality increases as with health risks of exposure to pollution ($\partial H/\partial y$). Similarly, it increases as the efficacy of health or preventive care services ($\partial H/\partial m$) diminishes.

## 5.4 WTP Estimates

The survey did not yield workable data on preventive care expenditures of households. We did obtain *qualitative* statements regarding "active" and "passive" responses to air pollution. Active responses entailed a greater expenditure of time and effort, and included activities such as participating in demonstrations against air pollution, writing protest letters, shutting windows, etc. About 16% of the sample indicated that they at one time or another engaged in such active behavior, but it would be very difficult to assign a monetary value to those activities. We did not ask any questions regarding purchases of home air conditioners (car air conditioners are relatively rare), because - given the climate of the country - these would be purchased almost solely to relieve the harsh effects of summer heat and humidity. We also attempted to find out whether pollution affected residential mobility, but only 1.7% of respondents who had moved in the previous two years indicated that this was the major reason for changing residences. (A similar percentage indicated noise pollution as the prime reason for moving.) Of course, we have no idea how many former residents of Haifa have migrated out of the region for this reason. All of this left us with no alternative but to assume that only medical care budget reallocations matter in households' health production decisions.

Rewriting eq. (13) as $(\partial m/\partial H)$ C $(\partial H/\partial y)$,[19] we estimated the first term using conditional probabilities. First, specifying a logit model, we estimated the probability of at least one doctor visit during a two-week recall period prior to the interview, for each of three health states: h=0, healthy; h=1, having symptoms; h=2, having symptoms and respiratory diseases. All the other explanatory variables (except AV14, see below) are dichotomous. Medical services covered here include doctor visits (mostly at primary health clinics belonging to one of the health maintenance organizations, the so-called "sick funds") of the interviewee, spouse, and children.[20] Logit regressions were estimated for doctor visits, including *private* consultations (separately for respondents, spouses, and children).

Table 7

The variable representing pollution, AV14, indicates *measured*

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[19] Assuming the health production function enables us to write express it in terms of its inverse, m(H,y), namely, that the conditions of the implicit function theorem hold.

[20] In Israel almost all medical services are publicly provided, then, unless they actually sought private medical services, people are usually not fully informed of the out-of-pocket expenses. However, it is reasonable to expect that they would take cognizance of the time and psychological costs involved in a clinic visit or a hospital stay. These may bear some relationship to the real economic costs of providing the service. Children visits to a physician refer to at least one visit by at least one child from the respondent's family, since children were *not* individually identified in the questions relating to health conditions. See also footnote 15 above.

(actual or extrapolated) $SO_2$ concentrations (in ppb).[21] The variable AV14 is significant in every regression.[22] Respondents with respiratory system problems are more inclined to seek medical help, and so are females, respondents with no children in the 0-18 age group (probably a proxy for older respondents), and those of Asian-North African origin (may also be related to belonging to a lower income group). The results for spouses and children were similar, with AV14 figuring in all of them, but they have not been used here.

Next, we specified a multinomial logit model to describe the relationship between health state and pollution levels, where $p_1$ = prob($h \geq 1$), and $p_2$ = prob($h=2$). The results are given in Table 8. Again, as expected from the discussion in Section 2 above, AV14 is highly significant. The coefficients of the socioeconomic variables have also the expected sign.

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Table 8
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Viewing the medical care use probabilities as conditional probabilities given one's health state, we have calculated the change - at mean values of the other explanatory variables - of reducing mean AV14 by 50% (going from $y_0$ to $y_1$. Viz.,

$$\Sigma_i \text{ p(doctor visit in past 2 weeks } / \ h_i) \times p(h_i/y=y_0)$$

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[21] Since pollution data is measured only at a few points in the Haifa metropolitan region (and only $SO_2$ on a continuous basis), it was necessary to extrapolate ambient concentrations for the rest of the survey neighborhoods using an *ad hoc* dispersion model. Average concentrations were computed for two-week periods preceding the date of any given interview. The two-week averages are based on half-hour concentration readings.

[22] An alternative set of regressions was run with the variable MAX14, representing maximum daily concentration for the preceding two-week period, but AV14 turned out to be a better predictor.

$- \Sigma_i$ p(doctor visit in past 2 weeks / $h_i$) × $p(h_i/y=y_i)$, $i=0,1,2$

The decrease amounted to 2.26% percentage points, or about 8% from present usage levels. Converting this result to expected number of annual visits, and multiplying by C, the cost per visit of NIS 30,[23] yields a *rough approximation* of WTP of NIS 32.43.

Of course, this figure is an underestimate: (a) It does not include visits of spouse and children; (b) it is based on a question which asked whether there was *at least* one visit during the preceding two-week recall period, but did not ask for the actual *number* of visits; (c) it does not include hospitalization cost[24] or medication costs[25] ; (d) finally, as explained above, it overlooks the labor cost savings.

An altogether different question is associated with the nature of medical care services in a country like Israel, where most of the population is covered by one form or another of a subsidized quasi-public health insurance scheme. In this sense individuals do not have to make budget reallocation adjustment in the way assumed in the model. However, as remarked above, time and inconvenience associated with a visit to a primary health clinic might nevertheless be playing a major role, not much different from that of money expenditures. This of course is another major drawback of the empirical results, but we surmise that CVM valuations may have well been similarly affected.

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[23] Although no statistics are available, we believe this figure to be close, though somewhat lower than the corresponding cost of a private consultation visit to a general practitioner.

[24] Respondents were also asked about hospitalization during the 12 month period preceding the interview for illnesses connected with the respiratory system, but the number of responses was too small for any meaningful analysis.

[25] The expected decrease in the probability of obtaining medication resulting from pollution reduction, has been calculated to reach 17% approximately (a decrease from p=0.113 to 0.094).

## 6. COST OF ILLNESS (COI) VALUATIONS

### 6.1 Consumption of Medical Services and Bed Day Losses

The COI approach normally covers direct (expenditures on medical services) and indirect (income reduction due to work day and productivity losses). As observed above, given that work loss has been neglected in the household production model, we have made an attempt to estimate these losses. Since individuals would not directly suffer the consequences of work loss days because of the almost universal coverage by employer-paid sick-day leave, this cost is distinctly a *social* cost. We would not expect it to be expressed through *individual* WTP valuations.

A binary response model was used to analyze bed days during the two week recall period. The response variable, STY, was defined as follows:

$$STY = \begin{cases} 1 & \text{if respondent missed one or more days} \\ 0 & \text{otherwise.} \end{cases}$$

Although our sample was large (n=954), the results are nevertheless based on a *relatively* small number of observations, since only 65 cases were respondents who reported that they were absent from work for at least one day during the fortnight. A model was fitted with both socioeconomic and health attributes, using backwards elimination to fit the logistic regression. The estimated equation is given in Table 9.

Table 9

When AV14 is reduced by 50%, the probability of at least one bed day decreases from p=0.051 to 0.041, a drop of 18 percent. Work loss days at present pollution levels constitute about 1.85% of all work days. The total expected annual savings in number of work loss days due to pollution abatement, $\Delta L$ (assuming 300 working days per year), is given by $\Delta L = E \times 300 \times 1.85 \times \Delta p$, where E is the number of employed persons (above age 15) in the metropolitan region, and $\Delta p = 0.18$. A similar calculation was performed for the *non*-working persons in the sample. The weighted mean sample percentage of bed days (corresponding to the working group's work loss days) is 3.57.

Assigning a money value to these savings, would of course vary with the specific assumptions relevant in each case. The present calculations were based on 1987 gross wages per salaried employee, including social benefits, of NIS 1,832 per month, or $1,221 (Central Bureau of Statistics, Statistical Monthly, April, 1988). At this wage rate, the money value of the savings would total NIS 10 million per year for the working group. For illustrative purposes, if we also value a day of a non-working person at 1/2 that of a working person, an additional savings of almost NIS 8.5 million would be achieved, for a total of NIS 18.5 million. On a *per household* level, the expected savings would amount to about NIS 185.0

## 7. COMPARATIVE EVALUATIONS

### 7.1 CVM vs. Indirect Approaches

Several writers (e.g., Randall, 1987; Mitchell and Carson, 1989) have noted that the CVM approach deals with *ex ante* valuations, while the indirect approaches are usually associated with *ex post* valuations. This implies that one therefore should *not* expect to necessarily obtain close estimates in the two approaches; but the opposite is not necessarily true, either. Reliability of either approach (*which* one would supposedly be an empirical question) might be questioned, however, if results derived from the *same* set of observations turn out to be vastly different. Hence, a comparison of the results from the various approaches should be illuminating. Table 10 summarizes the values obtained under the different approaches.

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Table 10

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The closeness of the valuations is quite encouraging. Although the indirect approaches cover all respondents, including zero bidders, it is assumed that the this approach yields true valuations of protest bidders as well, and hence, the comparison should be made with the true bidders (non zero and true zero) of the corresponding CVM experiments (Tables 3 and 4). It should be noted especially that the mean values of individual household valuations in the two approaches are within the same order of magnitude (NIS 9.8 vs. 34.5 for $WTP^c$, and 73.3 vs. 68.6 for $WTP^e$).

### 7.2 Health Production, COI and CVM valuations

Although very tenuous assumptions were made in applying the household health production approach, one observes the closeness of the results to the CVM valuations. Since the model measures responses to reduction in pollution, the appropriate comparison is with the $WTP^c$ valuations. Indeed, if other health and preventive care components were added, the results of the $WTP^c$ comparisons could have turned out to be even closer.

Theoretically, the cost of illness estimates should have at best provided a lower bound on WTP valuations. But this should *not* have been the case in the present study, given that COI estimates refer to *social* rather than individual WTP, and include components which do not figure directly in the individual's decision making process. Thus, households do not directly bear all the cost of air pollution damages. They are covered by medical insurance, and do not bear the full cost of medical services. Part of the premium is paid by employers and, furthermore, medical services are subsidized by the government. In addition, paid sick-leave is almost universal for salaried workers. But, moreover, people clearly do not possess the kind of dose-response information which would have enabled them to fully assess the economic impact of exposure and disease. These facts would necessarily be reflected in WTP valuations. One should also note that cost of illness estimates are probably more susceptible that the others to data "manipulation". The results are sensitive to what we assume about

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the appropriate values for work loss of employed and unemployed individuals, the ratio between privately purchased and publicly provided prescriptions, and the cost of physician visits, etc.

In a certain sense, one might speculate that CVM responses represent willingness to pay to reduce the direct *disutility* associated with morbidity, plus maybe the aesthetic disutility of air pollution. Namely, CVM valuations are essentially the psychological costs associated with pollution. Indeed, results presented elsewhere (Zeidner and Shechter, 1988) indicate that WTP is sensitive to anger and anxiety caused by perceived exposure to air pollution. If this were indeed the case, then the CVM valuations, or at least part of them, should be *added* to cost of illness valuations!

### 7.3 Some concluding comments

Within the framework of a study dealing with the valuation of benefits from pollution abatement, several approaches were investigated. A notable feature of the present study has been the use of the an *identical* data base - households, their attributes and responses - in all three approaches. While contingent valuation relies exclusively on direct question techniques, so that survey data are a *sine qua non*, market demand systems are normally estimated from aggregate, secondary market data. In this study, however, the same *primary* data base was used. Valid comparable valuations pertaining to the same set of households were thus obtained. Since all approaches are presumed to measure the same thing(s), one should *a priori* expect the results to be close.

In this vein, we view the results as rather encouraging and believe that they provide further impetus for the use of CVM. Of course, improved statistics on health and preventive care should offer an improved basis for alternative, indirect appaoraches.

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Table 1 Exposure to air pollution and morbidity in adults and children: Odds ratios and 95% confidence intervals

| Symptom or disease | Odds | Lower | Upper |
|---|---|---|---|
| | Ratio | Confidence limit | |
| A. Respondents | | | |
| Winter cough & cold | 1.432202 | 0.973711 | 2.106581 |
| Permanent cough without cold | 1.718250 | 1.267392 | 2.329494 |
| Winter cough without cold | 1.434010 | 1.151725 | 1.785482 |
| Permanent phlegm & cold | 1.572290 | 0.979119 | 2.524816 |
| Winter phlegm & cold | 1.400047 | 0.977708 | 2.004823 |
| Permanent phlegm without cold | 1.347164 | 1.024256 | 1.771871 |
| Winter phlegm without cold | 1.282863 | 1.040762 | 1.581281 |
| Cough & phlegm | 1.566648 | 1.247142 | 1.968010 |
| Winter phlegm & cold | 1.574363 | 1.252040 | 1.979665 |
| Wheezing & cold | 1.358134 | 1.052843 | 1.751950 |
| Wheezing while breathing | 1.352105 | 1.065619 | 1.715611 |
| Dyspnoea | 1.802163 | 1.530547 | 2.121982 |
| Rhinitis | 1.305185 | 1.043520 | 1.632463 |
| Eye "infection" | 1.302482 | 1.030010 | 1.647031 |
| Headache | 1.595975 | 1.356035 | 1.878371 |
| B. Children | | | |
| Cough or phlegm & cold | 1.695780 | 1.209402 | 2.377761 |
| Cough or phlegm without cold | 1.922437 | 1.365540 | 2.706449 |
| Cough or phlegm | 1.969765 | 1.528539 | 2.538353 |
| Wheezing with cold | 1.694218 | 1.150931 | 2.493961 |
| Wheezing | 1.466871 | 1.130402 | 1.903492 |
| Asthma or bronchitis | 1.487382 | 1.139776 | 1.940998 |
| Pneumonia | 1.269068 | 1.008890 | 1.596343 |
| Rhinitis | 1.271813 | 1.013503 | 1.595958 |
| Eye "infection" | 1.495645 | 1.109688 | 2.015841 |

Table 2. Willingness to pay equations - nonzero bids only

| Explanatory variable | Regression coefficients<br>WTP$^c$ | <br>WTP$^e$ |
|---|---|---|
| Demographic and socioeconomic variables: | | |
| Age (years) | -7.86 (0.29) | -0.73 (0.073) |
| Sex (1=female) | 55.28 (4.76) | |
| Education (years) | 12.18 (0.71) | |
| Blue collar worker (1=blue collar) | -53.33 (6.80) | |
| Number of children ages 0-18 | -24.64 (2.19) | -6.93 (1.56) |
| Ethnic origin I (1=born in Africa/Asia) | -26.93 (6.20) | |
| Ethnic origin II (1=born in Europe) | 109.38 (6.38) | |
| Annual municipal taxes | | 0.22 (0.006) |
| Attitudional variables: | | |
| Perceived exposure to pollution at work (1 = yes) | 81.29 (5.29) | 14.42 (4.31) |
| Perceived neighborhood air quality (1-6) | -21.14 (1.51) | |
| Believes budget share allocated to pollution abatement too high | -382.22 (57.85) | 101.72 (38.87) |
| Believes budget share allocated to pollution abatement too low | 163.85 (5.90) | |
| Ready to devote time to public activities concerned with pollution abatement (1=yes) | 39.62 (1.64) | 5.54 (1.30) |
| Perception of government influence on pollution abatement (1=yes) | -26.99 (5.90) | |
| Pollution induces defensive actions by respondent (1=yes) | | 8.63 (4.48) |
| Health status | | |
| Perceived health status (1=not healthy) | -67.55 (5.46) | |
| Family history (exc. respondent) of asthma, pnuemonia, or bronchitis (1=yes) | 24.60 (4.78) | 8.81 (4.29) |
| Family history (exc. respondent) of respiratory system symptoms (1=yes)** | 55.91 (4.58) | |
| Adjustment factor | -952.98 (23.63) | * |
| Intercept | -708.53 | * |
| Adj. $R^2$ | 0.54 | 0.64 |

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* Not significant.

** Cough, sputum, wheezing, dyspnoea

Table 3. CVM Experiments: WTP$^c$ (in NIS, per household, excluding protest zero bids, except in binary choice)

| Elicitation method | | N | Mean | Median |
|---|---|---|---|---|
| Sample | | 2,518 | 34.5 | |
| Standard max. WTP | | 1,855 | 37.7 | |
| Repeat bids: | One-time payment | | | |
| | 1st bids | 343 | 26.4 | |
| | 2nd bids | 195 | 67.8 (+22.2) | |
| | Annual payment | | | |
| | 1st bids | 343 | 26.4 | |
| | 2nd bids | 195 | 67.8 (+22.2) | |
| Binary choice | | 360 | 66.2 | 65.0 |

Table 4. CVM Experiments: $\text{WTP}^e$ (in NIS, per household, excluding protest zero bids except in binary choice)

| Elicitation method | N | Mean | Median |
|---|---|---|---|
| Sample | 1,704 | 68.6 | |
| Standard max. WTP | 1,348 | 70.9 | |
| Repeat bids: One-time payment | | | |
| 1st bids | 199 | 64.2 | |
| 2nd bids | 195 | 89.0 (+24.8) | |
| Annual payment | | | |
| 1st bids | 157 | 54.5 | |
| 2nd bids | 163 | 77.9 (+23.4) | |
| Binary choice | 360 | 69.1 | 67.2 |

Table 5. Direct (CVM) valuations of *perceived* air quality changes (Includes zero bids)

| Present pollution level | Pollution level *after* change | | | |
|---|---|---|---|---|
| | Good | Moderate | Poor | Very poor |
| Good | | (a) $\underline{WTP^e}$<br>Mean = 26<br>Median= 15<br>N = 847 | | |
| Moderate | (b) $\underline{WTP^c}$<br>Mean =37.9<br>Median=28<br>N =750 | | (c) $\underline{WTP^e}$<br>Mean = 40<br>Median= 28<br>N =749 | |
| Poor | | (d) $\underline{WTP^c}$<br>Mean =47.2<br>Median=40<br>N = 192 | | (e) $\underline{WTP^e}$<br>Mean =42.7<br>Median=32<br>N =192 |

* Values in table refer to means and medians of the indicated sample air quality stratum, and stated in NIS per household per year.

Significance Levels:

Nonparametric median test for 2 samples:

$H_0$: $WTP^c$ (cell b) = $WTP^c$ (cell d).................. 0.015

$H_0$: $WTP^e$ (cell a) = $WTP^e$ (cell c) = WTP (cell e).. 0.001

Paired t-test for means (2 tailed):

$H_0$: $WTP^c$ (cell b) = $WTP^e$ (cell c).................. 0.001

$H_0$: $WTP^c$ (cell d) = $WTP^e$ (cell e).................. 0.049

Table 6. Parameter Estimates of the Budget Share Equation*

| Parameter | Estimate | Parameter | Estimate |
|---|---|---|---|
| $\alpha_1$ | -0.348<br>(-110.38) | $\phi_{11}$ | 0.0006<br>(1.11) |
| $\gamma_1$ | -0.721<br>(-15.74) | $\phi_{12}$ | -0.0009<br>(-0.73) |
| $\gamma_2$ | -1.404<br>(-16.42) | $\phi_{13}$ | 0.004<br>(3.27) |
| $\beta_{11}$ | -0.181<br>(-21.06) | $\phi_{14}$ | 0.00002<br>(0.04) |
| $\beta_{12}$ | 0.039<br>(2.49) | $\phi_{15}$ | 0.0024<br>(2.12) |
| $\beta_{22}$ | -0.159<br>(-4.90) | | |
| $\delta_{11}$ | -0.417<br>(-12.16) | $R^2 = 0.27$ | |
| $\delta_{12}$ | 0.001<br>(0.06) | $N = 2,239$ | |
| $\delta_{22}$ | -0.527<br>(-8.77) | | |

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* Asymptotic t statistics in parentheses.

Table 7. Estimated Logit Regression: Consumption of Medical Care Services (Physician Visits) - Respondents

| Explanatory Variable | Regression coefficient | Standard error |
|---|---|---|
| Intercept | -4.397 | 0.217 |
| Health status | 0.715 | 0.097 |
| AV14 | 0.018 | 0.005 |
| Sex (1=female) | 0.405 | 0.134 |
| No children 0-18 yrs. (1=none) | 0.588 | 0.134 |
| Birth origin Asia-Africa (1=yes) | 0.346 | 0.154 |

n = 3,612

$\chi^2 = 125.5$ (5 df).

Dependent variable: 1 = visited a physician in past 2 weeks

Health status 0 = healthy
1 = suffers from at least one of symptom
2 = suffers from at least 1 disease

Table 8. Estimated Logit Regression: Health Risks and Exposure to Pollution - Respondents

| Explanatory Variable | Regression coefficient | Standard error |
|---|---|---|
| Intercept ($h_1$) | 0.880 | 0.134 |
| Intercept ($h_2$) | -0.732 | 0.134 |
| AV14 | 0.011 | 0.002 |
| Education (1=low level, 0-8 yrs.) | 0.248 | 0.078 |
| Birth origin (1=Europe or America) | 0.285 | 0.072 |
| Sex (1=female) | 0.289 | 0.064 |
| No children 0-18 yrs. (1=none) | 0.254 | 0.088 |
| Age of respondent (<40) | -0.845 | 0.120 |
| Age of respondent (41-50) | -0.481 | 0.123 |
| Age of respondent (51-60) | -0.372 | 0.102 |

n = 3,612

$\chi^2 = 316.5$ (8 df).

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Dependent variable:

$h_1$ = suffers from at least 1 symptom *or* disease

$h_2$ = suffers from at least 1 disease

Table 9. Restricted activity or bed days

| Explanatory Variable | Regression coefficient |
|---|---|
| AV14 | 0.028<br>(0.012) |
| Income (1="low" income-below NIS 1,300/mo.) | 0.80<br>(0.295) |
| Intercept | -3.79 |

$\chi^2 = 24.2$ (18 df).

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Dependent variable: 1 = Stayed home at least 1 day during the past two weeks.

Table 10. Comparisons Between Direct & Indirect Valuations[*]
(Including zero bids. Mean household values in NIS)

| | $WTP^c$ | $WTP^e$ |
|---|---|---|
| CVM | | |
| Standard bids | 37.70 | 70.90 |
| Repeat bids | 67.80 | 89.00 |
| Binary choice | 66.20 | 69.10 |
| Indirect | | |
| Expenditure function | 9.81 | 73.25 |
| Health production | 32.43 | |
| Cost of illness (bed days) | 185.0 | |

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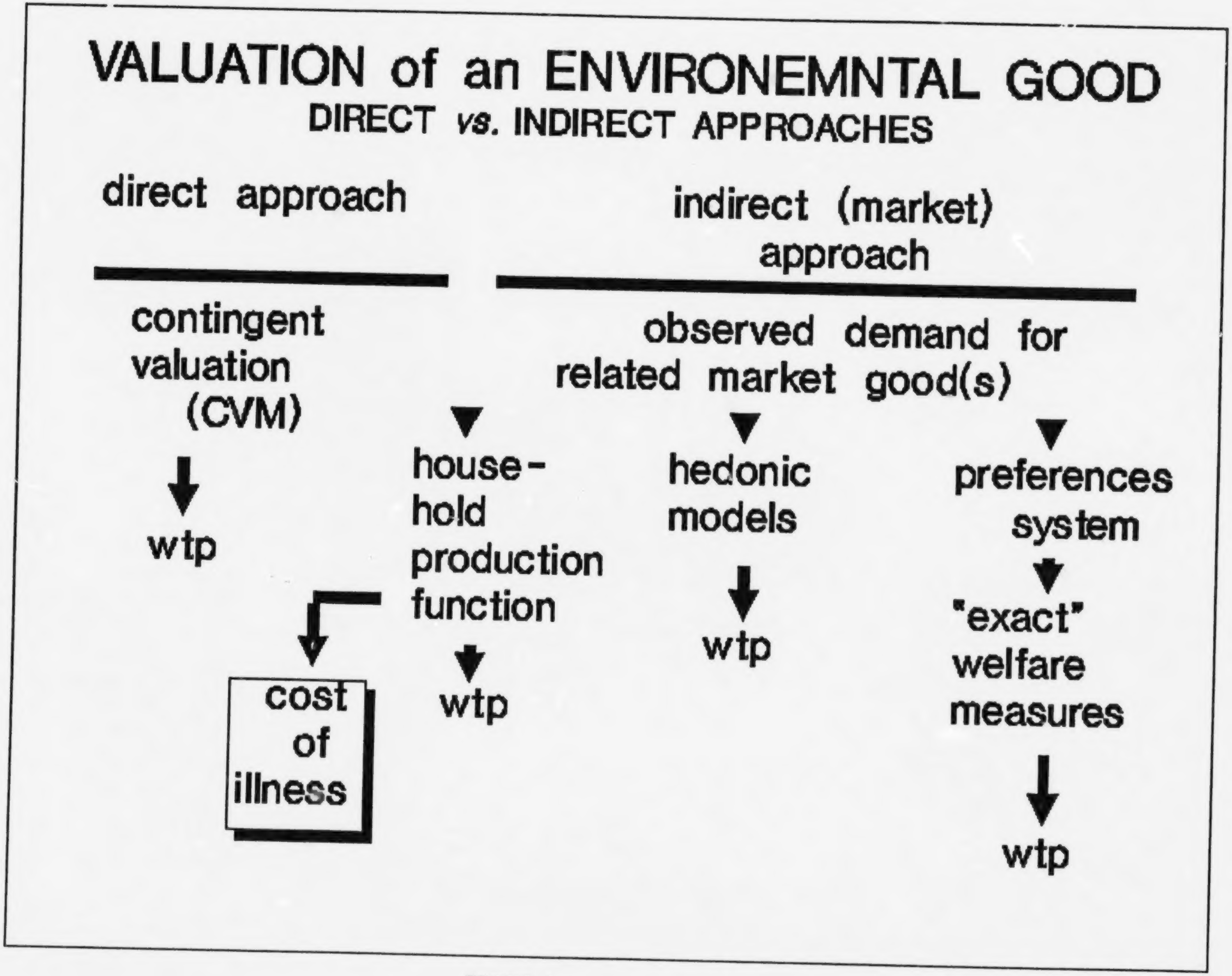
[*] Corresponding to changes in *perceived* pollution levels.



# VALUATION of an ENVIRONEMNTAL GOOD

## DIRECT *vs.* INDIRECT APPROACHES

direct approach

contingent valuation (CVM)

wtp

cost of illness

indirect (market) approach

observed demand for related market good(s)

household production function

wtp

hedonic models

wtp

preferences system

"exact" welfare measures

wtp

FIGURE 1

# ODD RATIOS FOR SYMPTOMS & DISEASES

( interviewee, children )

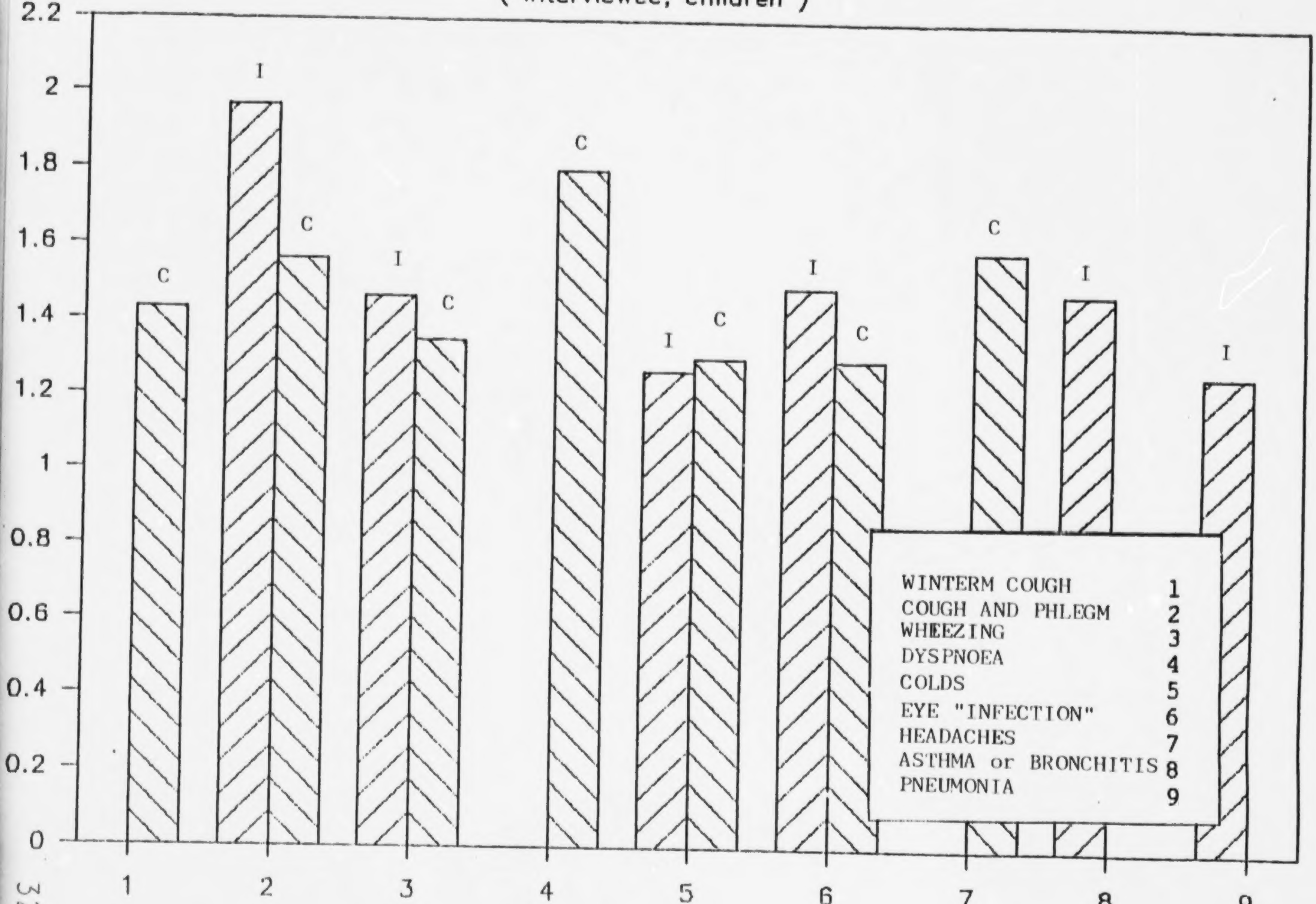


FIGURE 2

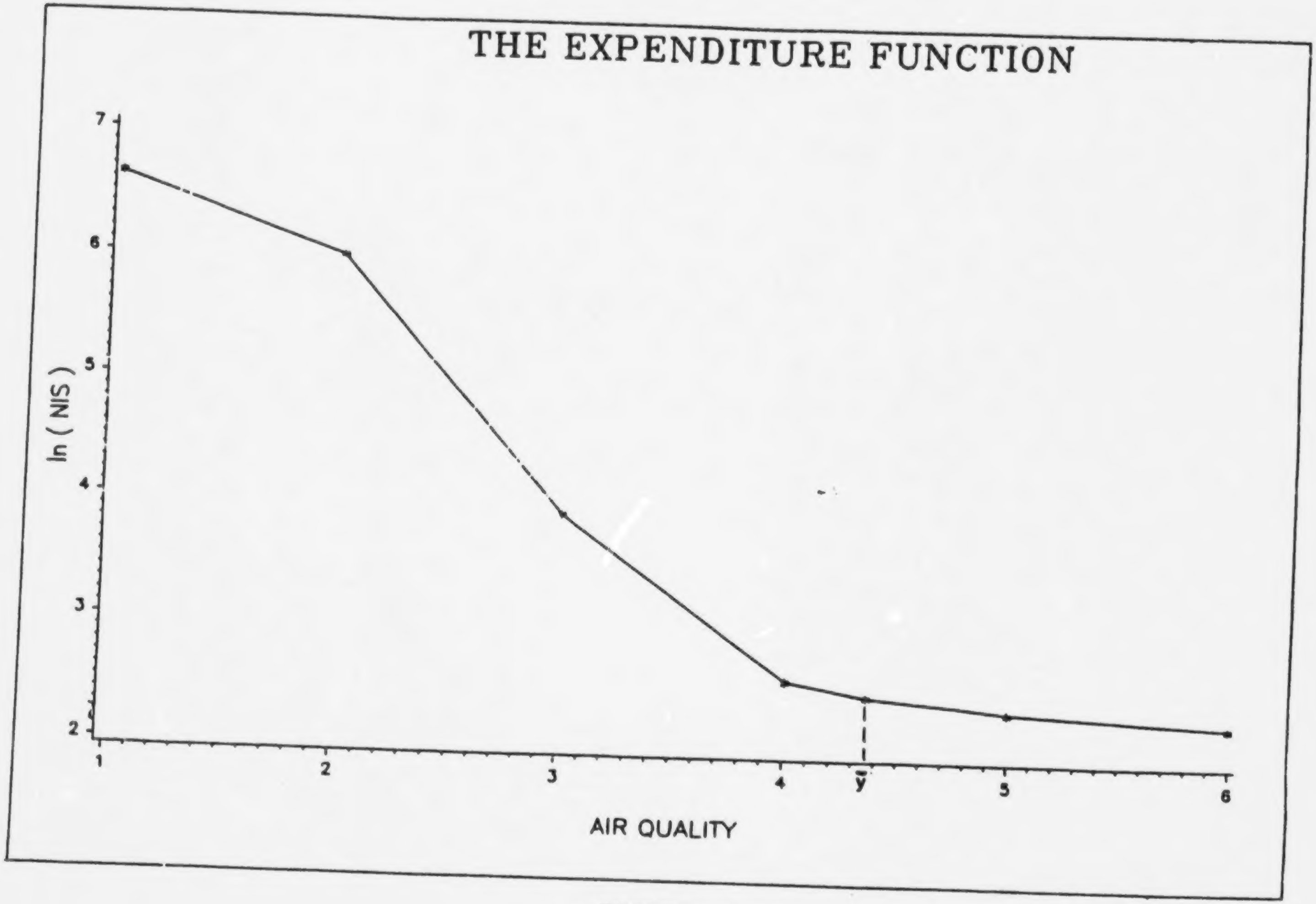

THE EXPENDITURE FUNCTION
ln ( NIS )
7
6
5
4
3
2
1
2
3
4
$\bar{y}$
5
6
AIR QUALITY

FIGURE 3

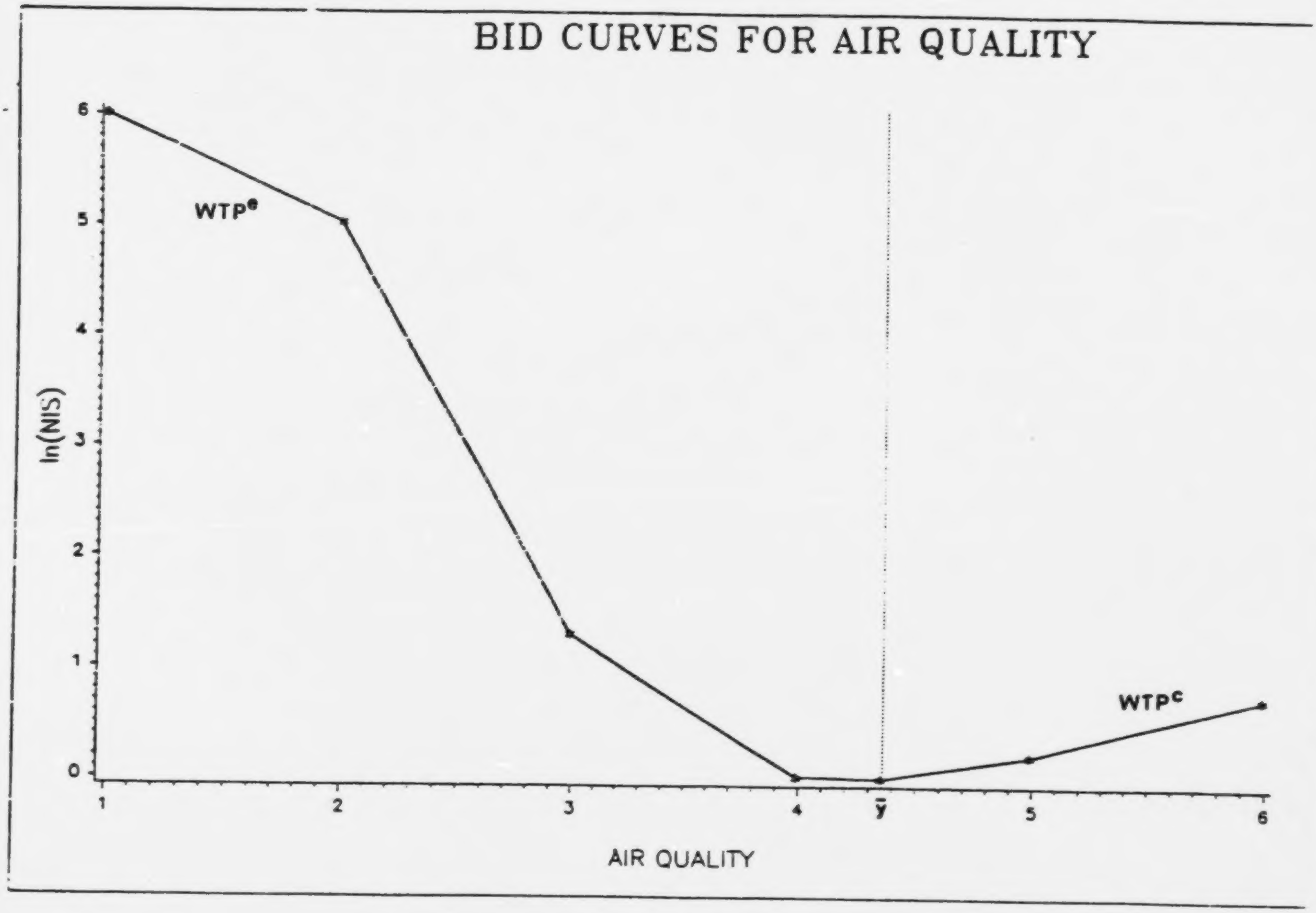

BID CURVES FOR AIR QUALITY
ln(NIS)
6
5
4
3
2
1
0
WTP$^e$
WTP$^c$
1
2
3
4
$\bar{y}$
5
6
AIR QUALITY

FIGURE 4

# Risk,
# Self-Protection
# and
# Ex Ante Economic Value*

by

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## Abstract

We examine the impact of self-protection on the ex ante value of reduced human exposure to an environmental hazard. Assuming a continuous distribution of health outcomes and self-protection that influences both the probability and the severity of an undesired outcome, we develop three propositions:

1) If risk is endogenous such that self-protection influences the probability or the severity of an undesirable outcome, then unobservable utility terms cannot be eliminated from the individual's ex ante valuation expression.

2) If risk is endogenous, knowledge of the convexity or the nonconvexity of physical dose-response relations is insufficient to sign unambiguously the change in an individual's ex ante marginal valuation of risk, even when consumer cognition is perfect.

3) If risk is endogenous, self-protection expenditures will not be a consistent lower bound of the ex ante value that a risk-averse individual attaches to a reduction in risk.

These three statements imply that several propositions originally developed for cases of exogenous risk and which form the analytical basis for most recent empirical work on the value of health risk changes are not immediately transferable to settings where endogenous risks prevail.

## I. INTRODUCTION

Any person who might suffer harm from exposure to an undesirable state of nature can reduce expected ex post costs by purchasing market insurance. Moral hazard, however, compels insurers to defray only a fraction of these costs [Arrow (1963), Shavell (1979)].[1] Consequently, individuals use self-protection to reduce both the ex ante probability and expected costs of the uninsured event [Ehrlich and Becker (1972)].[2] We consider the implications of this for models used to value risks to human health.

In particular, we find that:

1) Given moral hazard, when self-protection influences the probability, the severity, or both of an undesirable state, unobservable utility terms cannot be eliminated from the individual's ex ante valuation expression. Consequently, empirical studies that attribute differences across groups in ex ante value estimates solely to unobserved differences in household health production technologies are misplaced.

2) with moral hazard and self-protection, knowledge of the convexity or nonconvexity of physical dose-response relations is insufficient to sign unambiguously the change in an individual's ex ante marginal valuation for a reduction in the level of the hazard, even when consumer cognition is perfect. Therefore, we do not support the traditional argument that those individuals exposed to greater risk with greater income must place a higher value on a given risk reduction.

3) with moral hazard, an increase in the level of the environmental hazard does not necessarily lead to an increase in the level of self-protection. Therefore, self-protection expenditures are not a

consistent lower bound of the ex ante value a risk averse individual attaches to a reduction in risk.

These three statements imply that several propositions originally developed for cases of exogenous risk and which form the analytical basis for most recent empirical work on the value of health risk changes are not immediately transferable to settings where endogenous risks prevail.[3]

Berger, et al. (1987) appear to be among the first to consider endogenous risks in the context of human health.[4] Our treatment differs from their seminal effort in two significant ways. First, though they state the general continuous distribution case of risks to human health, they examine ex ante value only in a world of two mutually exclusive and independent states of nature: survival or death. We extend the ex ante value concept to the general continuous case. By maintaining continuity throughout, we allow the individual to choose between contractually defining states of nature or making an effort to alter states of nature. Spence and Zeckhauser (1972) demonstrate that the ability to influence states of nature enhances both the ex ante and the ex post gains from adaptation. In particular, we assume that individuals recognize that outcomes are stochastically related to actions, implying that predictions of behavior and the relative values that motivate it depend not only on preference orderings over outcomes, but also on preference orderings of lotteries over outcomes.

Second, Berger, et al. (1987) model only probability-influencing self-protection. They disregard the severity of the health outcome being risked, even though they concede that prior self-protection can influence both probability and severity. As pointed out by Ehrlich and Becker (1972) the distinction between self-protection that influences probability and self-protection that influences severity is somewhat artificial. The distinction is often said to be made for theoretical convenience [see for example Hiebert (1983)]. In contrast, we model the effects of self-protection that influences both the probability and the severity of the undesired state, and consider the effects on the ex ante value of reduced risk.

## 2. THE MODEL

Consider an individual who is involuntarily exposed to a health risk under a particular liability regime. Assume the risk is created by exposure to an ambient concentration of an environmental hazard, r, taken from the real interval, R:

$$R = [\underline{r}, \bar{r}] \tag{1}$$

Because of moral hazard, the individual cannot acquire enough market insurance to avoid the risk completely. The individual must decide from a real interval, S how much self-protection, s, to undertake:

$$S = [\underline{s}, \bar{s}] \tag{2}$$

Given exposure to the hazard, the individual is uncertain as to which, i, of N alternative health outcomes will occur. Let

$$H = \{h_1, h_2, \ldots, h_N\} \tag{3}$$

denote the outcome space where outcomes are the individual's human health capital returns ordered from smallest to largest, given the individual's genetic and development history.

Let $f(h_i; s, r)$ denote the probability of outcome i occurring given that self-protection, s, is undertaken and that the exposure level to the environmental hazard is r. Assume the following about $f(\cdot)$:

Assumption 1: $f(h_i; s, r) > 0$ for every $i \in [1, \ldots, N]$ and every $s \in S$ and $r \in R$.

Let $F(h_i; s, r)$ denote the corresponding distribution function defined over the support $[a, b]$

$$F(h_i; s, r) = \int_a^b f(h_i; s, r)dh \tag{4}$$

where a and b are the minimum and maximum health outcomes.[5] We assume the following about $F(\cdot)$:

Assumption 2: $F(h_i; s, r)$ is twice continuously differentiable in $s \in S$ and $r \in R$ for every $i \in [1, \ldots, N]$.

Assumption 3: $F_s(h_i; s, r) \leq 0$ for every $s \in S$ and $r \in R$ and every $i \in [1, \ldots, N]$ in the sense of first-order stochastic dominance.[6]

Assumption 4: $F_r(h_i; s, r) \geq 0$ for every $s \in S$ and $r \in R$ and every $i \in [1, \ldots, N]$ in the sense of first-order stochastic dominance.

Assumption 5: No restrictions are placed on the convexity of the distribution function in the immediate neighborhood of an optimal level of self-protection, $s^*$, for all $s \in S$ and $r \in R$ and for every $i \in [1, \ldots, N]$.

The individual is risk averse with a von Neumann-Morgenstern utility index over wealth W, U(W). The following assumptions are made about U(W):

Assumption 6: U is defined over the real interval $(\bar{W}, \infty]$ where $\bar{W}$ is 0.

Assumption 7: $\lim_{W \to \bar{W}} U(W) = -\infty$.

Assumption 8: U is strictly increasing, concave, and thrice continuously differentiable.

For each health outcome the individual might realize, he selects a minimum cost combination of medical care and foregone work and consumption. Let

$$C = C(h_i; s, r) \tag{5}$$

be his ex ante expectation of realized costs which depend on the uncertain health outcome, self-protection, and the exposure level to the hazard. Assume the following about C(•):

Assumption 9: C is strictly decreasing, convex, and thrice continuously differentiable in $s \in S$ for every $i \in [1, \ldots, N]$ such that $C_s < 0$ and $C_{ss} > 0$ for all $h \in H$.

Assumption 10: C is strictly increasing and thrice continuously differentiable in $r \in R$ for every $i \in [1, \ldots, N]$ such that $C_r > 0$. No restrictions, however, are placed on $C_{rr}$ and $C_{sr}$ for all $h \in H$.

Given incomplete insurance purchases, intertemporally separable utility, and constant expected prices for medical care, the individual's choice problem is then

$$\underset{S}{\text{Max}} \left[\int_a^b U(W - C(h;\, s,\, r) - s)dF(h;\, s,\, r)\right]. \tag{6}$$

Note that the price of self-protection has been normalized to unity. The subscript i is suppressed to maintain notational simplicity.

Given the model, we are now able to develop the propositions stated in the introduction.

## 3. EX ANTE VALUE AND WILLINGNESS-TO-PAY

3.1 Endogenous Risk. A few recent refinements to the willingness-to-pay approach to valuing environmental hazards have acknowledged the frequently endogenous form of the problem. For example, Rosen (1981), Berger, et al. (1987), and Viscusi, et al. (1987) note that self-protection affects survival or injury probabilities, while Shibata and Winrich (1983) and Gerking and Stanley (1986) allow self-protection to influence the severity of ex post damages. In a nonstochastic world or in an uncertain world with only two feasible states, these studies demonstrate that marginal willingness-to-pay can be expressed solely in terms of the marginal rate of technical substitution between hazard concentrations and self-protection. This result cannot be generalized to a continuous world with endogenous risk.

Proposition 1: Given the model assumptions, when self-protection influences either the probability or the severity of health outcomes or both, the individual's marginal willingness-to-pay for reduced risk cannot be expressed solely in terms of the marginal rate of technical substitution between ambient hazard concentrations and self-protection. In particular, unobservable utility terms cannot be eliminated from expressions for the ex ante value of reduced risk.[7]

Proof: To show that for a continuous distribution the individual's compensating variation statement of willingness to pay for reduced risk includes the unobservable utility terms, we examine self-protection that influences either the distribution or the severity (costs) of the health outcomes or both.

First, maximize the expected utility index (6) by selecting an optimal level of self-protection $s \in S$ yielding the following first-order condition for an interior solution

$$EU_w = -E[U_w C_s] + \int_a^b U_w C_h F_s \, dh. \tag{7}$$

The left-hand side of (7) represents the marginal cost of increased self-protection in terms of the utility of foregone wealth. The right-hand side reflects two types of marginal self-protection benefits: the first term is the direct utility effect of enhanced wealth resulting from reduced expected ex post costs; the second term is the indirect utility effect of a stochastically dominating change in the distribution of health outcomes.

The indirect effect was derived by integrating by parts the effect of self-protection on the distribution

$$\int_a^b U(\cdot)dF_s(\cdot) = UF_s\Big|_a^b + \int_a^b U_w C_h F_s dh$$

$$= \int_a^b U_w C_h F_s dh,$$

since $F_s(a;\cdot) = F_s(b;\cdot) = 0$. Assume that improved health outcomes will decrease the ex post costs, $C_h < 0$.

Solve for the compensating variation statement of the willingness-to-pay for reduced risk by totally differentiating the expected utility index (6), and then applying the first-order condition (7). When self-protection influences both the probability and severity of health outcomes such that $F_s < 0$ and $C_s < 0$, the willingness to pay expression is:

$$\frac{dW}{dr} = -\left[\frac{\int U_w C_h F_r dh - \int U_w C_r dF}{\int U_w C_h F_s dh - \int U_w C_s dF}\right] > 0, \tag{8}$$

where all integrals are evaluated over the support [a, b]. Obviously, the unobservable utility indexes cannot be removed from the individual's willingness to pay expression (8).

Even the assumption of a simple two state world fails to remove the utility terms from (8). For example, let $\pi(s, r)$ and $(1 - \pi(s, r))$ respectively represent the subjective probabilities of healthy and of sick states. Let $U_0(W - s)$ and $U_1(W - s - C(s, r))$ be the expected utility of being healthy or sick, where $U_0 > U_1$. The individual thus chooses $s \in S$ to maximize

$$EU = \pi(s, r)U_0(W - s) + (1 - \pi(s, r))U_1(W - s - C(s, r)). \tag{9}$$

Following the same steps as before, the willingness to pay expression is

$$\frac{dW}{dr} = -\left[\frac{\pi_r[U_0 - U_1] - (1 - \pi)U_1'C_r}{\pi_s[U_0 - U_1] - (1 - \pi)U_0'C_s}\right] > 0, \tag{10}$$

where $\pi_r < 0$, $\pi_s > 0$, $U_1' = \partial U_1/\partial W$, and $U_0' = \partial U_0/\partial W$. Again, utility terms cannot be removed.

Next allow, as do Gerking and Stanley (1986), self-protection to influence the severity, $C_s < 0$, but not the probability, $F_s = 0$, of health outcomes. Further assume that $F_r = 0$ which, with $F_s = 0$, implies that neither collective nor individual actions will influence the probability of a particular health outcome, i.e., hazard concentrations resemble sunspots or the phases of the moon. With these assumptions, expression (8) reduces to:

$$\frac{dW}{dr} = -\frac{E[U_w C_r]}{E[U_w C_s]} = -\left[\frac{EU_w EC_r - \mathrm{cov}(U_w,\ C_r)}{EU_w EC_s - \mathrm{cov}(U_w,\ C_s)}\right] > 0. \qquad (11)$$

For the unobservable utility terms to be absent from (11), the two covariance expressions must be zero; however, our model assumptions do not allow them to be zero. Therefore the two utility terms cannot be removed.

Finally, assume, as does Rosen (1981), that self-protection affects probability, $F_s < 0$, but not severity, $C_s = 0$. In Rosen's (1981) terms, one cannot be more severely dead. For similar reasons, $C_r = 0$. Under these conditions, expression (8) reduces to:

$$\frac{dW}{dr} = -\frac{\int U_w C_h F_r dh}{\int U_w C_h F_s dh}, \qquad (12)$$

and again the willingness-to-pay expression cannot be rid of the unobservable utility terms, which concludes the proof.*/

We could examine additional cases. For example, self-protection might influence only the probability of a health outcome, but hazard concentrations could affect probability and severity, or vice versa. The results would not change: utility terms would loom up in the willingness-to-pay expressions, implying that policy efforts to aggregate across individuals and to account simultaneously for the reality of probability and severity unavoidably involve interpersonal utility comparisons.

3.2 Nonconvex Dose-Response Relations. Proposition 1 poses hurdles to procedures which would establish a social risk-benefit test by summing unweighted compensating or equivalent variations across individuals.[9] Yet another problem for consistent aggregation is the ambiguous effect that a change in hazard concentrations has on the sign of compensating variation. In a contingent valuation study of the risk valuations attached to hazardous waste exposures, Smith and Desvousges (1986, 1987) report increasing marginal valuations with decreasing risk. This finding is but the latest in a 15-year long series of analytical [Starett (1972), Winrich (1981)] and empirical [Crocker (1985), Repetto (1987)] papers which use prior information on physical dose-response relations, individual abilities to process information about these relations, or individual perceptions of the relations to produce a declining marginal valuation result for more of a desirable commodity. However, when risk is endogenous, no one has yet asked whether convexity of the marginal value of risk follows when cognition is not an issue.

An individual's compensating variation can be shown to be ambiguous in sign even if the strongest possible case for negative effects of increased hazard exposure is imposed. To illustrate, define strong convexity as follows. Definition 1: Strong convexity of risk is defined as: convex ex post cost, $C_{rr} > 0$; convexity of the distribution function, $F_{rr} > 0$; and declining marginal productivity of self-protection, $C_{sr} > 0$, $C_{hr} > 0$, $C_{sh} > 0$ and $F_{sr} > 0$. Strong nonconvexity describes the conditions most favorable for the traditional argument that increased risk requires progressively increasing compensation to maintain a constant level of expected utility. Increased exposure increases the probability and the expected ex post costs of undesirable health outcomes to the hazard at an increasing rate; moreover, the marginal productivity of self-protection is decreasing across the board.

The opposite case is strong nonconvexity. Strong nonconvexity defines the weakest case for negative effects of increased exposure to the hazard.

Definition 2: Strong nonconvexity of risk is defined as: nonconvex ex post cost, $C_{rr} < 0$; concavity of the distribution function, $F_{rr} < 0$; and increasing marginal productivity of self-protection, $C_{sr} < 0$, $C_{hr} < 0$, $C_{sh} < 0$ and $F_{sr} < 0$.[10]

The following proposition states the result:

Proposition 2: Even in the absence of cognitive illusions or failure to consider all scarcity dimensions of the risk-taking problem, a maintained hypothesis of strong convexity of risk is insufficient to guarantee that increased exposure to a hazard requires progressively increasing compensation to maintain a constant level of expected utility. Similarly, strong nonconvexity is insufficient to guarantee progressively decreasing compensation.

The proposition is supported by Dehez and Drèze (1984, p. 98) who show that the sign of the marginal willingness-to-pay for safety given an increase in the probability of death is generally ambiguous. Drèze (1987, p. 172) concludes that any assertions about this sign given a change in safety "...must be carefully justified in terms of underlying assumptions".

Proposition 2 contradicts the argument of Weinstein, et al. (1980) and others that individuals at greater risk must have a greater demand for safety. Consequently, contrary to Rosen (1981), individuals at greater risk with greater wealth cannot necessarily be weighted more heavily when risk reductions are valued. Similarly, the assertions by Kahneman and Tversky (1979) and Smith and Desvousges (1987) that increasing marginal willingness-to-pay for reduced risk constitutes a lapse from rational economic behavior are not supported.[11]

Proof: To demonstrate that an increase in hazard concentration has an ambiguous effect on an individual's compensating variation, differentiate the compensating variation in expression (8) with respect to the hazard exposure:

$$\begin{aligned}\frac{d(dW/dr)}{dr} = &-\frac{1}{\Omega}\Big[E[U_{ww}C_r^2 - U_wC_{rr}] - 2\int[U_{ww}C_rC_h - U_wC_{hr}]F_r dh \\ &\qquad + \int U_wC_hF_{rr}dh\Big] \\ &+ \frac{\Delta}{\Omega^2}\Big[E[U_{ww}C_sC_r - U_wC_{sr}] + \int[U_wC_{hr} - U_{ww}C_hC_r]F_s dh \\ &\qquad + \int[U_{ww}C_sC_r - U_wC_{sr}]F_r dh + \int U_wC_hF_{sr}dh\Big],\end{aligned} \tag{13}$$

where

$$\Omega = \int U_wC_hF_s dh - \int U_wC_s dF > 0,$$

$$\Delta = \int U_wC_hF_r dh - \int U_wC_r dF < 0,$$

and all integrals are evaluated over the support [a, b].

The terms on the right-hand side of (13) can be defined in terms of direct and indirect utility effects given an increase in exposure to a hazard. $\Omega > 0$ and $\Delta < 0$ represent the combined first-order direct and indirect utility effects of s and r. The first and fourth terms in (13) represent second-order direct utility effects on expected costs with an increase in exposure. Given strong convexity, the sign of the first term is negative. The sign of the fourth term is ambiguous in the sense that alternative parameterizations are conceivable in which either $U_{ww}C_sC_r$ or $U_wC_{sr}$ dominates in absolute magnitude. The second, fifth, and sixth terms are second-order direct and indirect utility effects weighted by the marginal effect on the distribution of either s or r. Given strong convexity, the signs of all three terms are ambiguous in the above sense. Without prior information on the magnitude of the marginal effects on the expected cost function, there is no reason to expect one term to dominate. The third and seventh terms represent the second-order indirect and cross-indirect utility effects of increased exposure. By the definition of strong convexity, the sign on both terms is negative. Without knowing the relative magnitude of all the direct and indirect utility effects, however, strong convexity is insufficient to sign (13) unambiguously. Likewise, the assumption of strong nonconvexity is also insufficient to sign (13). Whether one imposes strong convexity or strong nonconvexity the sign of (13) is ambiguous. Although sufficient conditions for increasing or decreasing marginal willingness-to-pay can be determined, there is, in the absence of prior information or simple ad hoc assumptions, no reason to expect that one or two terms will dominate expression (13). This concludes the proof.

3.3 Self-Protection Expenditures as a Lower Bound. Consideration of self-protection has not been limited to problems of ex ante valuation under uncertainty. A substantial literature has emerged, e.g., Courant and Porter (1981), and Harrington and Portney (1987), which demonstrates that under perfect certainty the marginal benefit of a reduction in a health threat is equal to the savings in self-protection expenditures necessary to maintain the initial health state. This result cannot be extended to the uncertainty case when self-protection influences both ex ante probability and ex post severity.

Proposition 3: Neither strong convexity nor strong nonconvexity of risk is sufficient to sign the effect of a risk change upon self-protection expenditures. Therefore these expenditures cannot be used to determine the welfare effect of a risk change.

Proposition 3 contradicts Berger et al.'s (1987) argument that if increased exposure increases the marginal productivity of self-protection,

$F_{sr} < 0$, then self-protection will increase with exposure. Consequently, Berger, et al.'s (1987 p. 975) sufficient conditions for "plausible" results do not hold when self-protection influences both probability and severity.

Proof: To demonstrate that strong convexity is insufficient to determine the effect increased hazard exposure has on self-protection, take the first-order condition in equation (7) and apply the implicit function theorem. The effect of increased exposure on self-protection is

$$\frac{ds}{dr} = -\Big[E[U_{ww}C_r(1 + C_s) - U_wC_{rs}] + \int[U_wC_{sh} - U_{ww}C_h(1 + C_s)]F_r dh + \int[U_wC_{hr} - U_{ww}C_rC_h]F_s dh + \int U_wC_hF_{sr} dh\Big]/D \tag{14}$$

where

$$D = E[U_{ww}C_s(1 + C_s) - U_wC_{ss}] + 2\int[U_wC_{sh} - U_{ww}C_hC_s]F_s dh - \int U_{ww}C_hF_s dh + \int U_wC_hF_{ss} dh < 0 \tag{15}$$

and all integrals are evaluated over the support [a, b]. D is the second-order sufficient condition of the maximization problem (6), and is assumed to hold whenever (7) holds.

Given $D < 0$, the sign of (14) depends on the sign of its right-hand-side numerator. The first term in the numerator of (14) is the direct utility effect of increased exposure on expected costs. Given strong convexity of risk and $(1 + C_s) > 0$ from the first-order condition, the sign of the first term is negative. The second term reflects the indirect utility effect of increased exposure on the distribution. Given strong convexity, its sign is ambiguous in the earlier defined parameterization sense. The third term is a direct utility effect weighted by the marginal effect of self-protection on the distribution ($F_s < 0$), and its sign is also ambiguous. The signs for the second and third effect are ambiguous since there is no a priori reason to believe that any one set of terms dominates the others. The fourth term in the numerator is the cross-indirect utility effect of increased exposure. Given strong convexity, its sign is negative. Therefore, without prior information on the relative magnitudes of the four direct and indirect utility effects, strong convexity is insufficient to sign (14) unambiguously. Given the conditions most favorable to the traditional argument that increased risk will increase self-protection, we still require prior information on the impact that increased exposure has on the marginal productivity of self-protection to support the argument.

Following the logic above, an assumption of strong nonconvexity of risk leads to a similar conclusion of an ambiguous effect of increased exposure on self-protection. Consequently, since self-protection may decrease as exposure to a hazard increases, self-protection cannot be considered a consistent lower bound on the ex ante value a risk averse individual attaches to a reduction in risk. This concludes the proof.

## 4. CONCLUSIONS AND IMPLICATIONS

Individuals and policymakers use self-protection activities to influence both their ex ante risks and their expected ex post consequences. The implications of this for models used to value risks to human health are unequivocally negative. We show that unobservable utility terms cannot be eliminated from marginal willingness-to-pay expressions, implying that empirical efforts which identify marginal rates of substitution with willingness-to-pay are misdirected. We also show that even under the most favorable restrictions increased risk need not imply progressively increasing levels of compensation in order to restore initial utility levels. Consequently the traditional argument that those who are exposed to greater risk and have greater wealth must value a given risk reduction more highly does not follow. Finally, we demonstrate that increased risk need not imply increased self-protection expenditures; thus changes in these expenditures may not bound the value of a risk change.

Some succor for health risk valuation efforts could be obtained by stepping outside professional boundaries to draw upon prior information from psychology, biomedicine, and other disciplines. Insight might therefore be gained into the signs and the relative magnitudes of many terms in expressions (13) and (14). It is odd that the field of economics which explicitly recognizes the policy relevance of incomplete markets has historically been reluctant to use information from other disciplines in order to simulate the valuation results of a complete market. We recognize that there is a growing trend to incorporate restrictions drawn from other disciplines into the behavioral postulates of economic models.[12] The results of this paper suggest that the incorporation process should be accelerated.

Incorporation will not overcome, however, the aggregation problems posed by the presence of utility terms in individuals' willingness-to-pay expressions. Approaches to aggregate risk-benefit analysis do exist other than the mechanical summation of consumer surpluses calculated from the singular value judgement that social welfare and aggregate total income are synonymous. Given that individual consumer surpluses can be estimated, one possibility is to draw upon the extensive equivalence scale literature, e.g., Deaton and Muellbauer (1986), in order to weight each individual or household. Tradeoffs can then be evaluated using an explicit social welfare function which recognizes that personal health is in part self-produced and inalienable. Alternatively, utilities might be calculated directly.

## FOOTNOTES

1. Moral hazard refers to the tendency of insurance to influence an individual's incentive to prevent loss.

2. Self-protection includes everything from installing home water filters in order to reduce pollutant concentrations in drinking water to medical care and the use of tort law. [See Laffont (1980), Crocker (1984)].

3. The empirical human health valuation literature typically assumes that health risks are: (i) independent of individual actions; and (ii) usually for the sake of analytical and empirical tractability, individuals require progressively increasing levels of compensation to maintain constant expected utility when confronted by increasing risk. Jones-Lee, et al. (1985), for example, embodies both conditions. We argue these assumptions are unnecessarily restrictive in the sense that they stretch the ability of economic analysis to cover the domain of risky phenomena.

4. Psychologists agree that individuals perceive that they have substantial control over uncertain events [Perlmuter and Monty (1979)]. Stallen and Tomas (1984) conclude that "... the individual is not so much concerned with estimating uncertain parameters of a physical or material system as he is with estimating the uncertainty involved in his exposure to the threatening event and in opportunities to influence or control his exposure" [emphasis added].

5. The [a, b] interval could also be influenced in subsequent periods by self-protection. We disregard this issue.

6. Subscripts represent partial derivatives.

7. Assumptions of a risk-neutral individual with an identity map of ex post costs would eliminate the unobservable utility expressions. These assumptions seem excessively restrictive.

8. One might eliminate the utility terms by using the pointwise optimization technique that Mirrlees (1974) and Holmström (1979) employ. However, pointwise optimization evaluates self-protecting choices individually at each and every health state rather than in terms of lotteries over health states. It thus adopts an ex post rather than an ex ante perspective.

9. See Polemarchakis, et al. (1986) for thinking on aggregation under exogenous risk.

10. Rogerson (1985) assumes that the distribution function must generally satisfy the convexity of the distribution function condition (CDFC). Therefore, the assumption of a concave distribution in r and s is perhaps restrictive. As shown by Jewitt (1988), however, the CDFC assumption is not universally required in that it satisfies very few of the standard distributions set forth in statistics textbooks.

11. Close inspection of the questionnaire formats upon which these assertions are based reveals that respondent opportunities to influence risk and/or severity were not fully controlled.

12. See Wärneryd (1986), Weinstein and Quinn (1983) and Smith and Johnson (1988), for example.

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# THE ECONOMICS OF QUARANTINES: AN APPLICATION TO PESTICIDE REGULATION

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# THE ECONOMICS OF QUARANTINES: AN APPLICATION TO PESTICIDE REGULATION

One of the most common practices for dealing with hazardous situations is simply to remove the hazard from human proximity, either spatially or temporally. Such policies can be termed quarantines. The classic case is that of contagious disease control, where infected individuals are kept apart from vulnerable individuals until the threat of contagion has passed. Other examples include imprisoning dangerous criminals; locating hazardous industries (e.g., military testing grounds, nuclear power plants and other hazardous activities) in remote areas; keeping dangerous chemicals, high voltage equipment, etc. in locked or otherwise inaccessible locations; and keeping workers out of areas recently treated with pesticides.

Any quarantine involves tradeoffs that must be evaluated whether the decision maker is a government agency or an individual concerned with self-protection from self-generated hazards. The benefits of quarantines obviously consist of reductions in hazard. But quarantines typically have costs as well, such as additional discomforts and lost wages of contagious patients or productivity losses from suboptimal siting or scheduling. These tradeoffs must be evaluated in determining the appropriate parameters of a quarantine, that is, the length of time and/or location restriction. This paper develops a framework for optimal quarantine determination and applies it to a widespread form of quarantine, re-entry regulation of pesticide-treated fields. Section I contains a model of optimal quarantine determination. Section II models optimal timing of pesticide application under re-entry regulation. Interestingly, the imposition of re-entry regulation may make it optimal for farmers to switch to prophylactic treatment of pests, a practice which has been widely criticized as inefficient in the literature on pesticide use. Section III applies this model to the case of pre-harvest intervals in apple production in three major producing states. Section IV develops a model of acute poisoning from exposure to pesticide residues under different re-entry intervals. Section V combines the production and health models into a tradeoff model which is then used to obtain a rough evaluation of current policy.

## I. Optimal Quarantine Determination

Generally speaking, quarantine have both a spatial and a temporal dimension: how far away the hazard is sited and how long the quarantine lasts. Contagious disease quarantines have both: one must decide where to locate infectious patients relative to other patients and the general population as well as how long to continue isolation. Penal policy also does: prison location and length of sentence will both depend on how dangerous a criminal is. In other cases, one of these dimensions may be irrelevant. In pesticide regulation, for example, only the temporal dimension may matter: many pesticide residues are absorbed by touch and therefore the hazard affects only those entering a treated field. In siting of military testing grounds, nuclear power plants or other hazardous facilities, on the other hand, only location matters.

Let D represent the spatial dimension of the quarantine and T the temporal dimension. Let Z represent a consumption or production activity affected by the quarantine. The benefits of consumption or production, $B(Z,D,T)$, depend on Z and on the quarantine parameters D and T, as does the level of hazard, $H(Z,D,T)$. Let $W[B(Z,D,T), H(Z,D,T)]$ denote the utility function of an individual facing a hazardous situation or a social welfare function. The relevant decision problem is to choose Z, D and T to maximize utility or social welfare. This is typically accomplished in two stages. First, microeconomic theory is used to derive a model of optimal consumptive or productive behavior conditional on the quarantine parameters D and T. The resulting behavioral model is subsequently used to derive the optimal policy parameters.

Formally, letting subscripts denote derivatives, the necessary conditions are

(1a) $W_B B_Z + W_H H_Z = 0$

(1b) $W_B B_D + W_H H_D = 0$

(1c) $W_B B_T + W_H H_T = 0.$

The two-stage procedure described above consists of first solving equation (1a) to get the optimal level of consumption/production activity contingent on the quarantine, $Z^*(D,T)$, and then choosing D and T to maximize $W[B(Z^*(D,T),D,T), H(Z^*(D,T,),D,T)]$ according to the necessary conditions

(2a) $W_B(B_Z Z_D + B_D) + W_H(H_Z Z_D + H_D) = 0$

(2b) $W_B(B_Z Z_T + B_T) + W_H(H_Z Z_T + H_T) = 0.$

The case of pesticide regulation considered below is investigated by first deriving profit-maximizing pesticide use patterns conditional on temporal quarantine restrictions, $Z^*(T)$, and farm profits, $B(Z^*(T))$. The risk of acute organophosphate poisoning of farm workers is modeled as a function of pesticide use, $H(Z^*(T))$. These two components are combined into a tradeoff curve under an assumption of equal welfare weights on farm income, $B(Z^*(T))$, and worker safety, $H(Z^*(T))$, that is $W_B = W_H$. Finally, this tradeoff curve is used to derive the optimal length of the quarantine $T^*$ under different environmental conditions.

One can conceptualize distance-related quarantine problems in the same way.

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For example, the size, operating procedures and transmission line requirements of a nuclear power plant may depend on the distance between it and the population and industrial centers it serves, so that one would begin with a relationship between these factors and quarantine distance, $Z^*(D)$. The risks posed by the plant, $H(Z^*(D),D)$ depend on the quarantine distance D and the operating characteristics of the plant, $Z^*(D)$. These two can be combined using the appropriate welfare weights $W_B$ and $W_H$ to obtain a tradeoff relation that can then be used to determine the optimal distance $D^*$.

In sum, even in regulatory contexts it is typically necessary to solve private optimization problems prior to considering the social decision problem, since the private optimization problems are crucial elements of the tradeoff relations needed. Moreover, close interdisciplinary cooperation is often required to specify the hazard functions H, since they depend in complex ways on combined economic, environmental and biomedical factors.

## II. Crop Production Under Re-Entry Regulation

One of the most common measures used to protect farm workers and other rural inhabitants from the health hazards posed by applied pesticides is to forbid entry into treated fields for a specified period of time during which pesticide residue levels (and hence health risks) are thought to be excessive. Similar regulations aim to protect consumers as well by forbidding harvest for a specified interval after application of pesticides. Often, these re-entry regulations lead to reductions in growers' incomes by preventing optimal scheduling of harvest or intraseasonal activities like pruning or irrigation, causing decreases in yield, quality or price received for the crop. Thus, whether the decision maker is a government agency charged with protecting farm workers or a farmer deciding whether to work in his/her own field, the determination of an appropriate re-entry interval hinges on the choice of a tradeoff between risks to human health and safety, on the one hand, and the economic losses induced by regulation on the other.

For the sake of simplicity, we concentrate on the problem of re-entry regulations affecting an individual farmer's harvest of a perishable crop (fruits, vegetables), the kind of crop to which this form of regulation is applied most often. We assume that benefits B are restricted to farm profits, which are a function of pesticide use Z, itself a function of the re-entry interval T. We assume also that the farmer applies the pesticide at a standard application rate and focus on the determination of the timing of the application.

Assume that there is a time $t_0$ representing the earliest date at which the crop can be harvested; prior to $t_0$, the crop will be immature and hence not harvestable. Assume also that after $t_0$, the value of the crop declines because of decreased quality or because of price decreases due to seasonal increases in aggregate production, so that the farmer's revenue is maximized by harvesting at $t_0$. Formally, this implies a revenue function $R(t)$ such that $R(t_0) = \max \{R(t)\} = R^*$, and, letting subscripts denote derivatives, $R_t < 0$ and $R_{tt} \leq 0$ for $t > t_0$. Production costs, including pesticide materials and application costs, will be assumed to be constant and will thus be ignored.

Now assume that a pest appears at a time $t_a$ shortly prior to the optimal harvest time $t_0$. If left untreated, the pest will damage a proportion of the crop which will then be unsalable. The larger the pest population is, the greater the level of damage will be. This damage can be avoided by treating the crop with a pesticide. To simplify matters, assume that only a single standard treatment is available at a negligible cost. If the farmer treats the crop immediately upon arrival of the pest, i.e., chooses a treatment time $t_s = t_a$, the pest will be effectively eradicated and damage will be essentially reduced to zero. If, on the other hand, the farmer treats the crop before the pest arrives ($t_s < t_a$), the pesticide will decay; its effectiveness will be reduced by the time the pest arrives and the farmer will sustain some crop losses. The longer is the interval between treatment and the arrival of the pest, the greater will be the decay of the pesticide and the damage caused by the pest.

These characteristics can be represented formally by letting the proportion of the crop damaged by a pest population of size k be a function $g(k, t_a - t_s)$, where $t_a - t_s$ represents the time elapsed between treatment and the arrival of the pest. The preceding discussion suggests that $g_k > 0$, $g_t > 0$ and $g(k,0) = 0$. Pesticide decay curves are typically convex, so that one would expect $g_{tt} \geq 0$ as well.

There are two types of treatment strategies available to farmers: a reactive strategy of applying pesticides upon the arrival of the pest, and a prophylactic or preventive strategy of applying pesticides in anticipation of a pest problem. The reactive pest management strategy will maximize profits whenever it is feasible, which implies an optimal choice of $t_s = t_a$ whenever $T \leq t_0 - t_a$. If the re-entry period T is sufficiently long, however (specifically $T > t_0 - t_a$), following the reactive treatment plan may force the farmer to delay the harvest and thereby lose revenue. In this case the farmer faces a tradeoff between losing revenue from crop damage and losing revenue from harvesting delays. Under some conditions, it may become optimal for the farmer to adopt a prophylactic treatment strategy. While this practice has been much maligned in the pest management literature, rigidities is scheduling such as those imposed by re-entry regulation may make it desirable for farmers.

Some casual empirical evidence supports the notion that re-entry intervals actually provide a motivation for prophylactic treatment strategies. In Oregon, plum growers expecting to need to use parathion for end-of-season codling moth control typically apply the chemical 14 days -- the length of the pre-harvest interval -- prior to the projected harvest date, regardless of whether the pest is in evidence.

It should be clear that the farmer will never treat any earlier than needed to be able to harvest at time $t_0$, i.e., that $t_s \geq t_0 - T$; treating any earlier than $t_0 - T$ would imply accepting greater damage in return for no gain in revenue and is thus less profitable than treating at $t_0 - T$. It should also be evident that the farmer will always harvest the crop as soon as possible, that is, at least as soon as the re-entry period has ended. If the re-entry constraint is non-binding, then the harvest time will be $t_0$. If the re-entry constraint is binding, then the harvest will occur T periods after the treatment time; normalized (without loss of generality) to fit the revenue curve R. This can be written $t_s + T - t_0$.

The pesticide use patterns adopted and revenues earned by the farmer thus depend critically on whether or not the re-entry interval constitutes a binding constraint. If it does not, then a reactive treatment strategy is always optimal, $t_s = t_a$, the crop will be harvested at $t_0$ and revenue will be $R^*$. If it does, the farmer will face a tradeoff between crop damage and decreased revenue. The optimal pest management strategy will be determined by the choice of a treatment time $t_s$ which maximizes realized revenue, given by:

$$(3) \quad [1 - g(k,t_a - t_s)]R(t_s + T - t_0)$$

subject to the constraint:

$$(4) \quad t_0 - T \leq t_s \leq t_a.$$

Because the convexity of the pesticide decay function makes the damage function $g(k,t_a - t_s)$ convex, the realized revenue function (3) will be convex unless R is quite strongly concave. Thus, the optimal treatment plan must be analyzed according to two cases.

Case 1: The most likely case is that realized revenue (3) will be convex, so that the optimal treatment time will be either the maximum or minimum possible time, that is, either $t_a$ or $t_0 - T$. In essence, of course, this constitutes a choice between reactive ($t_s = t_a$) and prophylactic ($t_s = t_0 - T$) treatments. The farmer will choose the one which gives the greatest profit. If $t_s = t_a$, there will be no damage ($g = 0$) but the farmer will have to wait until $t_s + T - t_0$ to harvest and will thus realize a revenue of $R(t_a + T - t_0)$. If $t_s = t_0 - T$, there will be damage $g(k,t_a + T - t_0)$; the farmer will harvest at $t_0$ and thus realize a revenue $[1 - g(k,t_a + T - t_0)]R^*$. If the difference between these two realized revenues,

$$(5) \quad V = R(t_a + T - t_0) - [1 - g(k,t_a + T - t_0)]R^*$$

is positive, the farmer will adopt the reactive strategy and treat at $t_a$. If it is negative, the farmer will adopt the prophylactic strategy and treat at $t_0 - T$. An increase in the size of the pest population k will increase V and thereby make the farmer more likely to adopt a reactive strategy. An increase in the re-entry interval T, though, will increase V only if the marginal increase in the proportion of the crop damaged by treating earlier ($g_t$) is less than the marginal increase in the proportion of revenue lost by treating later ($R_t/R^*$). Thus, if $g_t > R_t/R^*$, an increase in T will make the farmer more likely to adopt a prophylactic strategy. An increase in the interval between the arrival of the pest and the optimal harvest data, that is, in $t_0 - t_a$, will, of course, have precisely the opposite effect of an increase in the re-entry interval T.

Case 2: If the revenue function R( ) is sufficiently concave to make realized revenue (3) concave, the profit-maximization problem will have an interior solution defined by:

$$(6) \quad g_t R + (1 - g)R_t = 0$$

with sufficiency assured by:

$$(7) \quad Q = g_{tt} R + (1 - g)R_{tt} \leq 0$$

which holds by assumption. It is readily apparent that an increase in the re-entry interval will lead the farmer to treat earlier $(dt_s/dT = -[R_t g_t + (1 - g)R_{tt}]/Q < 0)$, thereby accentuating the tendency toward prophylactic treatment. If, as one would expect, the increase in damage from treating earlier is greater for larger pest populations than for smaller ones (i.e., $g_{tk} \geq 0$), an increase in the pest population size will induce the farmer to treat later $(dt_s/dk = -[g_{tk} R - g_k R_t]/Q > 0)$, thereby reducing the tendency toward prophylactic treatment. As before, an increase in $t_0 - t_a$ will have the opposite effect of a increase in T.

## III. Pesticide Use in Apple Production

Consider the case of re-entry regulation of organophosphate insecticides used to protect apple crops from infestations of codling moth larvae from moth flights shortly prior to harvest. The yield and quality of the apples is assumed to increase up until the maturity date $t_0$, which is the earliest date at which the crops may be harvested. After $t_0$, yield and quality will remain constant for a considerable length of time. However, the price the farmer receives for the crop will decline as time passes because the aggregate supply of apples will increase as producers in other regions harvest and market their crops. This price decline will continue until the price of apples for fresh consumption equals the price for processing uses, at which point the price will remain constant. An analysis of the intraseasonal trends in farm-level apple prices in three major producing states (Washington, Michigan, California) indicated that this price decline is convex and could be represented well by an exponential curve. Thus, the price received by a grower harvesting a full crop at time $t \geq t_0$ is $R^*\exp(-a(t - t_0))$.

The threat posed by a late-season flight of codling moths consists of an infestation of larvae in the fruit, i.e., of wormy apples. This threat can be alleviated by using organophosphates to kill the moths before they lay eggs. Standard doses of these pesticides are typically applied; without loss of generality, normalize this standard dose to unity. Pesticide decay rates are typically modeled as exponential curves, so that the proportion of the pest population killed by a treatment applied at $t_s$ is $\exp(-b(t_a - t_s)$ and the proportion surviving is $1 - \exp(-b(t_a - t_s))$. Assume that all infested fruit is unsalable and that the proportion of the crop damaged is proportional to survivorship. Letting k represent the proportion of the crop damaged by a moth population of standard size, the damage function $g(k, t_a - t_s)$ will be in this case $k[1 - e(-b(t_a - t_s))]$.

The realized revenue function (3) in this case will thus be:

$$(8) \quad R = R^* \exp(-a(t_s + T - t_0)) \; (1 - k[1 - \exp(-b(t_a - t_s))])$$

which is obviously convex. The difference in profit between treating at $t_a$ and treating at $t_0$ is thus

$$(7)\quad V = R*\exp(-a(t_a + T - t_0)) - R*(1 - k[1-\exp(-(t_a + T - t_0))]).$$

which will be positive whenever

$$k > [1 - \exp(-a(t_a + T - t_0))]/[1 - \exp(-b(t_a + T - t_0))] = k_c$$

and negative whenever $k < k_c$. The optimal treatment strategy is thus:

$$(9)\quad t_s = \begin{cases} t_a, & k > k_c \\ t_0 - t, & k < k_c \end{cases}$$

In addition to the comparative static results from the general case it is straightforward to show that the faster the price declines over the season, the more likely the farmer is to adopt a prophylactic strategy ($dV/da < 0$) and that the faster the pesticide decays, the more likely the farmer is to adopt a reactive strategy ($dV/db > 0$).

To provide a empirical mechanism for evaluating the impact of re-entry regulation of pre-harvest use of parathion on apples in three main U.S. producing states (Washington, California, Michigan), the model was parameterized as follows. A regression of weekly data on farm-level prices received in Washington, California and Michigan over the period 1971-1980 on a time trend and dummies to control for differences among years and states yielded an estimate of the revenue decay parameter $a = 0.0024$. According to Johannes Joost, California extension specialist on apples, the maximum price received in 1984 was about $300/ton, which, at a yield of 10 tons/acre, suggests a maximum revenue of $150,000 for a 50-acre block. The regression analysis suggested that price levels in Michigan and Washington were about 17 percent and 32 percent above that of California; however, because Michigan harvests about 4 weeks after California and Washington, 2 weeks, the maximum price in these states should be 9.8 percent and 28.2 percent higher than California, respectively, giving estimates of about $165,000 per 50-acre block in Michigan and $192,000 per 50-acre block in Washington. An estimate of the parathion decay parameter $b = 0.8$ was taken from Spear et al.'s (1975a) study of parathion decay in California citrus orchards; examination of parathion decay data on Washington apples (Staiff et al. (1975)) indicated that the decay patterns in the two cases were essentially identical. Conversations with farm advisors indicated that, if left untreated, a codling moth infestation caused by a population of normal size would damage about 10 percent of the crop; thus, k was given a value of 0.10. Calculation of the damage threshold for prophylactic spraying over the range of reasonable re-entry periods, $k_c$, resulted in values ranging from .009 to .065, all well below k; thus, it appears that reactive treatment will always be optimal. In fact, apple prices would have to fall 2-10 times more rapidly before prophylactic treatment would become desirable.

## IV. Residue Poisoning From Parathion Exposure Among Apple Harvesters

The risk of clinical illness in workers as a result of exposure to residues of parathion applied to apples at various locations was modelled according to the overall scheme laid out by Popendorf and Leffingwell (1982). In essence, the pesticide is applied, a decay process takes place in which some of the parathion is converted to the oxygen analog, paraoxon, and exposure takes place days or weeks later when crews enter the field to harvest the crop. If clinical illness results, it is usually due to a dermally absorbed dose of paraoxon.

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There is considerable information available to quantify the various steps in this process but very limited data on climatological effects on the decay process itself.

The characterization of the residue decay process follows that of Spear et al. (1975a) and Popendorf and Leffingwell (1978). In both cases, the dislodgeable foliar residues of parathion and paraoxon are described by linear ordinary differential equations. The parameterization of these models utilized data obtained from citrus crops, but limited data on apples suggests a similar decay pattern (Staiff et al. (1975)). The simplified form of the model used here describes the residue relevant to worker hazard from day three post-application onwards. After day three the parathion residue has decayed to the point where the hazard to workers depends almost entirely on the paraoxon residue (Spear (1975b)).

The form of the model is:

$$dx/dt = -bx \tag{10a}$$

$$dr/dt = cx - qr \tag{10b}$$

where parathion residue is denoted by x and the paraoxon residue by r. The units are in $ng/cm^2$. The solution to this set of equations is:

$$x(t) = x_0 \exp(-bt) \tag{11a}$$

$$r(t) = (cx_0/b + q) [\exp(-qt) - \exp(-bt)] \tag{11b}$$

where t is the time post-application in days.

There are, then, four parameters required to solve for $r(t)$, the paraoxon residue, $b$, $c$, $q$, and the initial condition $x_0$. The first three parameters are weather dependent whereas the last depends on the application rates and pre-existing levels of foliar dust on the trees. Nigg et al. (1978) have studied the effect of weather variables on the parathion decay process and have concluded that rainfall and leaf wetness from other sources are the primary determinants of the rate of residue disappearance after the period immediately post application. Hence, climatological variability was modeled by assuming that the decay parameters, $b$, $c$, and $q$, are the same for all three regions but that the paraoxon residue is diminished as an exponential function of the cumulative rainfall during the decay period. Under these assumptions the rainfall-modified paraoxon residue at entry time $T$ is given by:

$$(12) \quad r'(T) = r(T) \exp(-.291CR)$$

where CR is the cumulative rainfall during the period $(0,T)$. A one inch rainfall leads to a diminution of the residue by 25 percent and a two inch rainfall a 44 percent decline. These predictions are more or less consistent with the data presented by Gunther et al. (1977).

Estimates of the parameters $b$, $c$ and $d$ are available from Popendorf and Leffingwell (1978). Also, the initial condition, $x_0$ was estimated from their data by regressing their parameter $a_0$ against the applied amount in pounds of active ingredient per acre (AIA). The resulting expression is:

$$(13) \quad x_0 = 1690(\mathrm{AIA})^{.3067} \ \mathrm{ng/cm^2}$$

The values used for the other parameters are $b = 0.8$, $c = 0.08$ and $q = 0.05$.

Following the procedure detailed by Popendorf and Leffingwell (1982) the dermal dose in mg/kg is related to the paraoxon residue by the expression $k_d r'(t) t_e$ where $t_e$ is the exposure time in hours and $k_d$ a constant determined empirically and set equal to 9.0 as observed in citrus crops. The exposure time is taken to be an eight hour shift. For a single organophosphate the relation between dermal dose and fractional inhibition of red blood cell cholinesterase (RBCD) is given by:

$$(14) \quad RBCD = 1 - \exp(-w_e D/LD_{50})$$

where, for paraoxon, the dermal $LD_{50}$ is 1.0 and $w_e$ equals to 6.0, midway in the reported range of 4.7 to 7.3. All members of a work crew are assumed to be exposed to the same residue environment which is further assumed to result in the same cholinesterase depression. Individual variability is modeled only in the relationship between cholinesterase depression and clinical illness.

The relationship between cholinesterase depression and clinical signs and symptoms of poisoning was modeled by assuming the probability of illness depended on the degree of cholinesterase depression according to the expression:

$$(15) \quad P = 1/[1 + \exp(w_1 + w_2 \, RBCD)]$$

where $w_1$ and $w_2$ were based on clinical experience and values reported in the medical literature (Midtling et al. (1985), Milby (1988)). Two sets of parameters were used, one relating to mild illness and the other to severe illness. The probability of illness relates to each member of the crew at the end of one eight-hour day and not to exposures cumulated over several days.

## V. Profit-Health Tradeoffs in Re-Entry Regulation

The models presented in the two preceding sections can be used to evaluate the impact of re-entry regulations on apple growers' revenues and apple harvesters' safety. The analysis was conducted under the assumptions that a flight of coddling moths arrives four days before the optimal harvest date $t_0$ (i.e., $t_0 - t_a = 4$), that parathion is applied at a rate of 2.0 pounds of active ingredient per acre, and that, as is typical, the crop produced on a 50-acre block will be harvested in one day by a crew of 500 (10 workers per acre). Losses in growers' revenues were compared to the risk of severe and mild poisoning to each individual worker. Rainfall levels of 0, 0.5, 1, 1.5, and 2 inches during the re-entry period were used to take into account the differences in weather conditions encountered in the different regions under investigation: California receives virtually no rainfall during the harvest period, Washington receives an average of 0.5 inches and Michigan receives an average of 1.5 inches under normal conditions.

Table 1 shows the expected numbers of severe and mild parathion poisoning cases under California, Washington and Michigan conditions, plus the fraction of revenue lost due to harvest delays. The risk of poisoning is clearly non-negligible: With a pre-harvest interval of four days or less, there will be an average of 2.5 severe cases and 43 mild cases under California conditions, 1.6 severe and 29 mild cases under Washington conditions and 0.8 severe and 15 mild cases under Michigan conditions. (At any given time, there will be almost 19 times as many mild as severe cases.) Each additional day entry is prohibited reduces the number of mild and severe cases by about 13 percent, while each additional inch of rainfall reduces them by about 75 percent. Even so, the risk of poisoning remains non-negligible for a relatively lengthy period of time: If re-entry is prohibited for as much as 2 weeks, there will still be an average of one severe poisoning incident for roughly every 2 50-acre blocks harvested in California, one severe incident for every 3 50-acre blocks harvested in Washington and one severe incident for every 4 50-acre blocks harvested in Michigan.

At the same time, the losses imposed by re-entry regulation can be considerable. Each additional day's delay in harvesting reduces total revenue by about 0.24 percent, corresponding to $360 per 50-acre block in California, $460 per 50-acre block in Washington and $395 per 50-acre block in Michigan. By way of contrast, total harvesting labor costs amount to about $425 per 50-acre block in Washington (Hinman, Tukey and Hunter). A pre-harvest interval of 2 weeks would result in a revenue loss on the order of 2.5 percent; since profit margins in Washington apple production range from 3 to 10 percent (Hinman, Tukey and Hunter), such a loss would represent a sizable fraction of net income.

The optimal pre-harvest interval in each state (assuming equal social welfare weights on farmers' incomes and workers' health) is determined by equating the marginal cost of additional harvest delays in terms of revenue lost with the marginal benefits associated with reductions in the number of poisoning incidents. For illustrative purposes, we calculated these optimal pre-harvest intervals under the conservative assumptions that benefits were restricted to average avoided costs, that is, to the average costs of hospitalization plus average lost wages. This ignores long-term losses due to chronic neurotoxic effects, the value of the disutility of suffering poisoning, losses caused by additional risks to consumers from residues remaining at the time of ingestion and so on.

A typical severe parathion poisoning case typically requires 3 days of hospitalization, with the first day spent in intensive care, followed by two weeks of recovery, i.e., lost work time. Assuming average costs of $1200 per day for intensive care and $500 per day for a standard hospital bed implies total hospitalization costs of $2200. Assuming an average wage of $10 per hour for an 8-hour day implies total lost wages of $800, for a total cost of $3000 per severe case (Becker (1988)).

A typical mild case requires no hospitalization; medical care will typically cost about $40 per case and there will generally be 2 days of lost work time, for a total cost of $200 per case (Becker (1988)).

Figures 1, 2 and 3 show the respective marginal costs and marginal benefits from severe and all poisoning cases associated with different pre-harvest intervals in California, Washington and Michigan. The optimal pre-harvest intervals are 15 days in California, 12 days in Washington and 9 days in Michigan. Current EPA regulations require 14 days regardless of rainfall conditions for applications of parathion on apples such as the one considered here. Interestingly, the current pre-harvest interval is quite close to the optimal levels calculated here, although our calculations suggest the desirability of greater conservatism under California conditions and less conservatism under Michigan conditions. They also suggest that, as long as local rainfall can be monitored effectively, the same levels of safety implicit in the 14-day pre-harvest interval can be achieved at lower cost by making the pre-harvest interval dependent on rainfall. For example, lowering the pre-harvest interval from 14 to 9 days when there have been 2 inches of rain would cut the losses suffered by Michigan apple growers by $1944 per 50-acre block, almost 50 percent, while lowering it from 14 days to 12 days when there have been 0.5 inches of rain would cut the losses suffered by Washington growers by $904 per 50-acre block, almost 20 percent.

## VI. Conclusions

Public authorities frequently use quarantines to ensure public safety by removing people from hazardous situations either in time or space. Individuals may pursue similar strategies to enhance their own safety in dealing with hazards. This paper develops a methodology for assessing the tradeoffs between productivity or utility losses from this type of regulation and reductions in risk of disease, accident or illness and applies it to the case of re-entry regulation in pesticides. We show that this form of regulation provides a rational incentive for prophylactic applications of pesticides, a practice that has been much maligned in the pesticide literature. In an empirical evaluation of pre-harvest intervals for parathion used on apples, we demonstrate that the tradeoffs involved are quite substantial, that the optimal pre-harvest intervals implied by rather conservative benefits estimates are quite close to those actually set by the Environmental Protection Agency, and that the same level of worker safety as that implicitly targeted by EPA can be achieved at lower cost by making pre-harvest intervals dependent on rainfall.

In order to focus on the main issues in deriving tradeoffs from quarantine parameter choices, the model used here is partial and rather stylized. Obvious improvements include incorporating considerations such as: pest population dynamics and intraseasonal effects; general equilibrium effects of re-entry regulation on prices and the distribution of production; choice of amounts of pesticides and harvest crew size as well as time of application; the influence of stochastic factors such as weather and size and time of arrival of pest populations; and uncertainties about residue decay, dermal absorption, cholinesterase depression and clinical response. The results we obtain, however, strongly suggest that more elaborate modeling of re-entry regulation and other forms of quarantine is well worthwhile.

Further research along these lines is especially necessary because environmental and occupational health problems such as the one addressed here are a growing policy concern. While policy advice has been monopolized by natural scientists until recently, recognition of the fact that absolute safety is often unattainable has led to an appreciation of the importance of evaluating tradeoffs between enhanced safety and other social goals. A key problem is that thorough tradeoff assessments require close interdisciplinary cooperation in modeling a full spectrum of economic, physical and biological processes beginning with production and terminating in risks to health.[1] While the difficulties of organizing such interdisciplinary cooperation have meant that this sort of modeling has been performed only seldom in the past, hopefully the work reported here will demonstrate the feasibility and importance of pursuing it.

## VII. Footnotes

[1] While economists have studied the links between pollution and health (as in the voluminous literature on air pollution and health initiated by Lave and Seskin) and between production and pollution (see for example, Anderson, Opaluch and Sullivan), to our knowledge none have modeled the entire path from production to pollution to health.

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# TABLE 1

## HEALTH RISKS AND REVENUE LOSSES UNDER ALTERNATIVE RE-ENTRY INTERVALS

| Re-entry interval (days) | Expected number of severe poisonings | | | Expected number of mild poisonings | | | Fraction of revenue lost |
|---|---|---|---|---|---|---|---|
| | California | Washington | Michigan | California | Washington | Michigan | |
| 0-4 | 2.46050 | 1.63800 | 0.81650 | 42.6950 | 29.2650 | 15.0000 | 0 |
| 5 | 1.95600 | 1.33250 | 0.69100 | 34.5800 | 24.0600 | 12.7600 | 0.002397 |
| 6 | 1.57650 | 1.09650 | 0.59100 | 28.2250 | 19.9600 | 10.9500 | 0.004788 |
| 7 | 1.28550 | 0.91250 | 0.51050 | 23.2450 | 16.7150 | 9.4850 | 0.007174 |
| 8 | 1.06000 | 0.76750 | 0.44520 | 19.3150 | 14.1300 | 8.2900 | 0.009554 |
| 9 | 0.88350 | 0.65250 | 0.39155 | 16.2050 | 12.0600 | 7.3050 | 0.011928 |
| 10 | 0.74500 | 0.56000 | 0.34725 | 13.7200 | 10.3850 | 6.4900 | 0.014296 |
| 11 | 0.63400 | 0.48540 | 0.31045 | 11.7300 | 9.0250 | 5.8100 | 0.016659 |
| 12 | 0.54550 | 0.42450 | 0.27965 | 10.1200 | 7.9100 | 5.2350 | 0.019016 |
| 13 | 0.47340 | 0.37450 | 0.25370 | 8.8050 | 6.9900 | 4.7555 | 0.021368 |
| 14 | 0.41470 | 0.33315 | 0.23165 | 7.7300 | 6.2250 | 4.3460 | 0.023714 |
| 15 | 0.36960 | 0.29865 | 0.21290 | 6.8400 | 5.5900 | 3.9965 | 0.026054 |
| 16 | 0.32645 | 0.26970 | 0.19680 | 6.1050 | 5.0550 | 3.6965 | 0.028389 |
| 17 | 0.29305 | 0.24530 | 0.18295 | 5.4850 | 4.5995 | 3.4380 | 0.030718 |
| 18 | 0.26500 | 0.22450 | 0.17095 | 4.9515 | 4.2130 | 3.2135 | 0.033041 |
| 19 | 0.24125 | 0.20680 | 0.16000 | 4.5245 | 3.8825 | 3.0185 | 0.035359 |
| 20 | 0.22110 | 0.19155 | 0.15135 | 4.1495 | 3.5985 | 2.8480 | 0.037672 |
| 21 | 0.20385 | 0.17840 | 0.14335 | 3.8280 | 3.3530 | 2.6980 | 0.039978 |
| 22 | 0.18900 | 0.16700 | 0.13635 | 3.5515 | 3.1400 | 2.5660 | 0.042280 |
| 23 | 0.17620 | 0.15705 | 0.13010 | 3.3120 | 2.9540 | 2.4495 | 0.044575 |
| 24 | 0.16510 | 0.14835 | 0.12460 | 3.1040 | 2.7915 | 2.3465 | 0.046866 |
| 25 | 0.15540 | 0.14070 | 0.11970 | 2.9230 | 2.6485 | 2.2545 | 0.049150 |
| 26 | 0.14690 | 0.13400 | 0.11535 | 2.7640 | 2.5225 | 2.1725 | 0.051430 |
| 27 | 0.13945 | 0.12805 | 0.11145 | 2.6245 | 2.4110 | 2.0995 | 0.053704 |
| 28 | 0.12835 | 0.12275 | 0.10795 | 2.5010 | 2.3120 | 2.0340 | 0.055972 |

qt-tab.wp/dlw/12/23/88

Figure 1
Optimal Re-Entry Interval in California

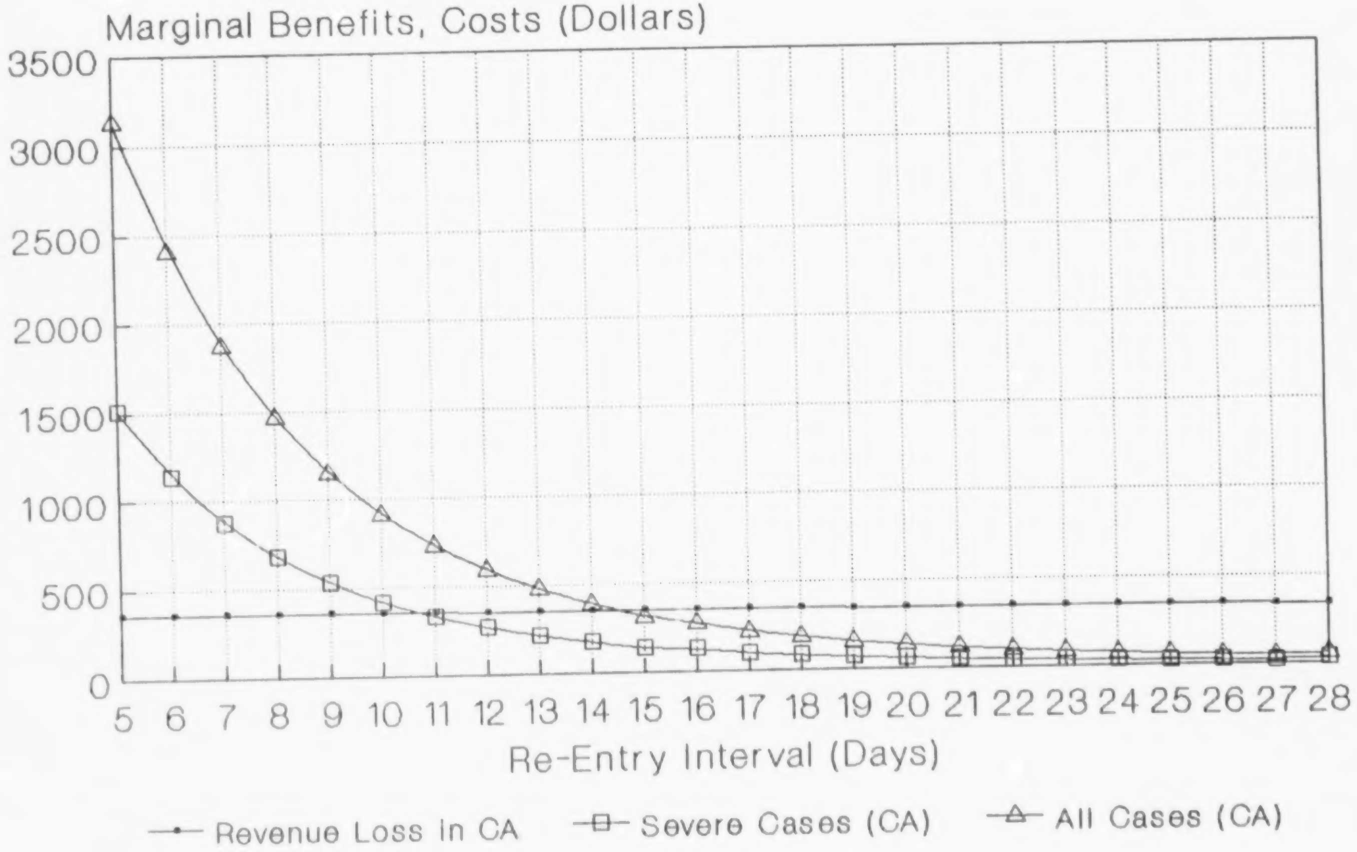
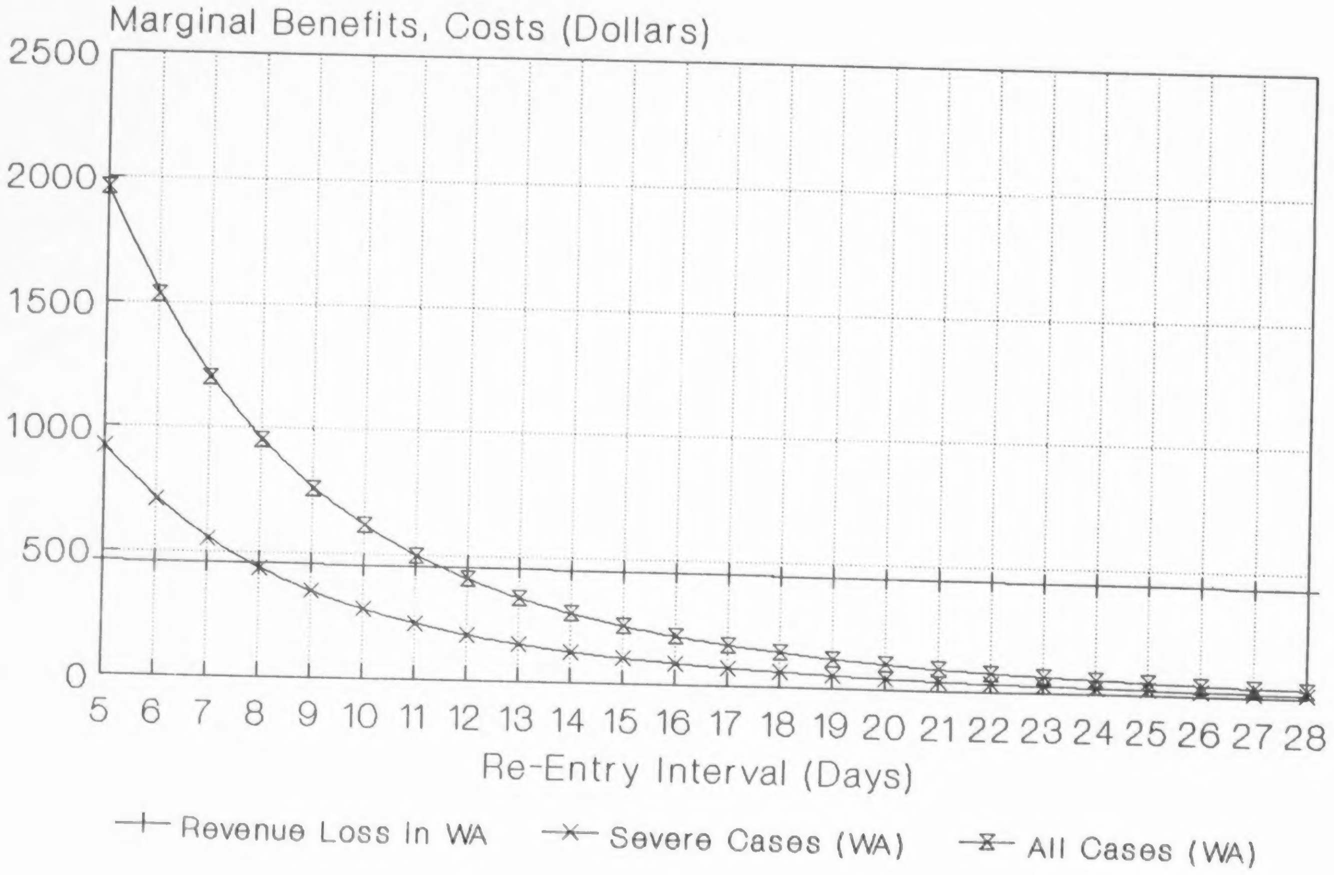
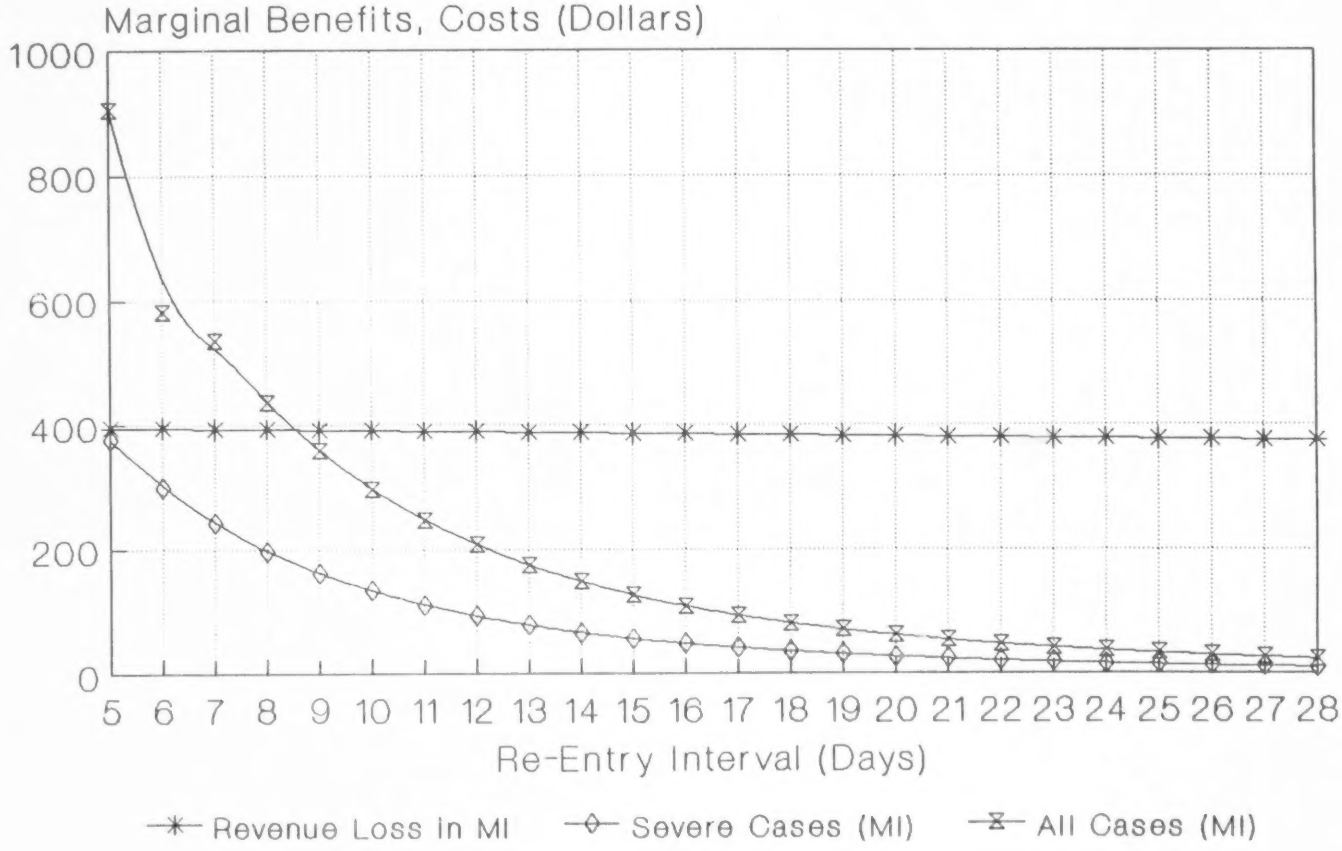


Figure 2
Optimal Re-Entry Interval in Washington



## Figure 3
## Optimal Re-Entry Interval In Michigan



# VALUING REDUCED MORBIDITY:
# A HOUSEHOLD PRODUCTION APPROACH*

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# ABSTRACT

This paper presents a unique application of the household production approach to valuing public goods and nonmarket commodities. Technical relationships are estimated between health attributes, private goods that affect health, and air quality using panel data drawn from a special survey. Statistical tests suggest that individuals equate marginal rates of technical substitution in household production with relevant price ratios. This result confirms that input choices are rational and is critical for estimating values of health attributes and air pollution. Value estimates obtained also bear on current questions facing environmental policymakers.

## I. Introduction

Individuals frequently apply a household technology to combine public and private goods in the production of nonmarket commodities for final consumption. Hori (1975) demonstrates that in these situations, market prices of private goods together with production function parameters may encode enough information to value both public goods used as inputs and nonmarket final consumption commodities. Although this valuation methodology is objective and market based, it seldom has been applied for three reasons. First, underlying technical relations either are unknown or data needed to estimate them are unavailable. Second, even if relevant technical information is at hand, the consumer's budget surface in commodity space may not be differentiable when joint production and other complicating factors are present. As a consequence, the commodity bundle chosen is consistent with any number of marginal rates of substitution between commodities and values of public goods and nonmarket commodities remain unknown. Third, joint production and nonconstant returns to scale also pose serious difficulties when taking the closely related valuation approach of estimating the area behind demand curves for private goods inputs and final consumption commodities (Bockstael and McConnell 1983). The problems posed by joint production are troublesome because Pollak and Wachter (1975) have argued that jointness is pervasive in home production, and Graham and Green (1985) found empirical evidence of substantial jointness in their estimation of a household technology.

This paper presents a unique application of the household production approach to valuing public goods and nonmarket commodities which allows for certain types of joint production and addresses key problems identified by previous authors. Technical relationships are estimated between health attributes, private goods, and air quality. Data used in the analysis are drawn from a special survey designed to implement the household production approach. Econometric estimates allow for censored dependent variables and cross-equation error correlations in panel data using tobit models with individual-specific variance components. Wilcox-Gök (1983, 1985) previously applied variance components estimation in a health context but did not examine censoring and cross-equation correlation. Key results of the present paper are: (1) attempts to value detailed attributes of nonmarket home produced commodities may be ill-advised; however, estimating a common value for a broadly defined category of attributes may be possible, and (2) statistical tests support the hypothesis that individuals equate marginal rates of technical substitution in household production with relevant price ratios. The latter result confirms that input choices are rational in the sense of Russell and Thaler (1985): choices are consistent with utility maximization subject to a correct understanding of the home technology. Also, value estimates obtained bear on current questions concerning air pollution control policy. The Clean Air Act of 1970 and its subsequent amendments focus primarily on health to justify regulation and require air quality standards to protect even the health of those most sensitive to pollution. The survey data are sufficiently rich to allow separate value estimates for persons with normal respiratory function and persons with chronic respiratory impairments.

The remainder of this paper is divided into four sections. Section II describes a simple household production model in a health context and reviews theoretical issues in obtaining value estimates. Section III discusses the survey instrument and the data collected. Section IV presents econometric estimates of production functions for health attributes, as well as values of better air quality and improved health for both the normal and respiratory impaired subsamples. Implications and conclusions are drawn out in Section V.

## II. Preliminaries

The model specifies utility (U) as a function of market goods (Z) and health attributes, called symptoms, (S).

$$U = U(Z, S) \tag{1}$$

For simplicity, Z is treated as a single composite good, but S denotes a vector measuring intensity of n health symptoms such as shortness of breath, throat irritation, sinus pain, headache, or cough. Intensity of the $i^{th}$ symptom is reduced using a vector (V) of m additional private goods that do not yield direct utility, a vector of ambient air pollution concentrations ($\alpha$), and an endowment of health capital ($\Omega$).

$$S^i = S^i(V, \alpha; \Omega) \qquad i = 1, \ldots, n \tag{2}$$

Elements of V represent goods an individual might purchase to reduce intensity of particular symptoms, and $\Omega$ represents genetic predisposition to experience symptoms or presence of chronic health conditions that cause symptoms. Notice that equation (2) allows for joint production in that some or all elements of V may (but do not necessarily) enter some or all symptom production functions.[1] The budget constraint is

$$I = P_Z Z + \Sigma_j P_j V_j \tag{3}$$

where $P_Z$ denotes the price of Z, $P_j$ denotes the price of $V_j$, and I denotes income.

Aspects of this general approach to modeling health decisions have been used in the health economics literature (e.g., Grossman 1972; Rosenzweig and Schultz 1982, 1983), where medical care is an example of V often considered. In these three papers, however, the stock of health rather than symptoms is treated as the home produced good, and Grossman treats decisionmaking intertemporally in order to analyze changes in the health stock over time. A multiperiod framework would permit a more complete description of air pollution's cumulative physiological damage, but the present model's focus on symptoms of short duration suggests that a one period model is appropriate. Moreover, long term panel data containing both economic and health information necessary to assess cumulative physiological damage are difficult to obtain.

Similar models also have been used in environmental economics to derive theoretically correct methods for estimating values of air quality and other environmental attributes (e.g., Berger et al. 1987, Courant and Porter 1981; Harford 1984; Harrington and Portney 1987). These models, however, only consider the case in which $m = n = 1$ and rule out the possibility of joint production. In this situation, the marginal value of or willingness to pay (WTP) for a reduction in air pollution can be derived by setting $dU = 0$ and using first order conditions to obtain

$$WTP_{\alpha} = - U_1 S^1_{\alpha}/\lambda = - P_1 S^1_{\alpha}/S^1_1 \qquad (4)$$

where $U_1$ denotes marginal disutility of the symptom, $S^1_{\alpha}$ denotes the marginal effect of air pollution on symptom intensity, $S^1_1$ denotes the marginal product of $V_1$ in reducing symptom intensity, and $\lambda$ denotes marginal utility of income. As shown, marginal willingness to pay to reduce symptom intensity $(- U_1/\lambda)$ equals the marginal cost of doing so $(- P_1/S^1_1)$.

Extensions to situations where m and n take on arbitrary values have been considered in the theory of multi-ware production by Frisch (1965) as well as in a public finance context by Hori (1975). Actually, Hori treats four types of household production technology. His case (3) involving joint production appears to best characterize the application discussed in Section IV because a single $V_j$ may simultaneously reduce more than one symptom. In this situation, a key result is that marginal values of home produced commodities cannot be re-expressed in terms of market prices and production function parameters unless the number of private goods is at least as great as the number of commodities ($m \geq n$). Intuitively, if $m < n$, the individual does not have a choice among some alternative combinations of symptom intensities because there are too few choice variables ($V_j$) and the budget surface on which each chosen value of $S^i$ must lie is not differentiable.[2]

Another perspective on this result can be obtained from the first order conditions of the individual's utility maximization problem. After substituting the symptom production functions into the utility function, the first order conditions include the budget constraint and

$$\begin{aligned} U_z - \lambda P_z &= 0 \\ \Sigma_i U_i S^i_j - \lambda P_j &= 0, \quad j = 1, \ldots, m. \end{aligned} \tag{5}$$

The marginal value of a reduction in air pollution is a weighted sum of the values of the individual symptom intensities ($U_i/\lambda$), where the weights are the marginal products of pollution ($S^i_\alpha$): $WTP_\alpha = - \Sigma_i (U_i/\lambda) S^i_\alpha$). Estimating values for reductions in symptoms or pollutants on the basis of observable behavior requires solving for the ($U_i/\lambda$) as functions of market prices of private goods and production function parameters. Rearranging the m first order conditions for the $V_j$ gives

$$\begin{bmatrix} S_1^1 & \cdots & S_1^n \\ \cdot & & \\ \cdot & & \\ \cdot & & \\ S_m^1 & \cdots & S_m^n \end{bmatrix} \begin{bmatrix} U_1/\lambda \\ \cdot \\ \cdot \\ \cdot \\ U_n/\lambda \end{bmatrix} = \begin{bmatrix} P_1 \\ \cdot \\ \cdot \\ \cdot \\ P_m \end{bmatrix} \tag{6}$$

If $m < n$, the rank of the symptom technology matrix $S = \{S_j^i\}$ is at most m and the system of equations in (6) is underdetermined. Intensity of one symptom cannot be varied holding others constant, and the marginal value of an individual symptom cannot be determined. On the other hand, if $m = n$ and the symptom technology matrix is nonsingular, then the rank is n and unique solutions can be computed for the $U_i/\lambda$. If $m > n$ and the technology matrix has full rank, then the system is overdetermined, and values for the $U_i/\lambda$ can be computed from a subset of the first order equations.

This theoretical overview yields several ideas useful in empirical application. First, if $m \geq n$ and the household technology matrix has rank n, then values of nonmarket commodities and public goods are calculated in a relatively straightforward manner because utility terms can be eliminated. Second, the possibility that $m < n$ suggests that the household production approach may be incapable of estimating separate values for a comparatively large number of detailed commodities and that aggregation of commodities may be necessary to ensure $m > n$.[3] Third, even if $m \geq n$, the household production approach may fail if there is linear dependence among the rows of the technology matrix. Thus, statistical tests of the rank of the matrix should be performed to ensure differentiability of the budget surface. Fourth, if $m > n$, first order conditions impose constraints on values that can be taken by the $S_j^i$; rejection of these constraints would imply that the outcome of the choice process is inconsistent with utility-maximization subject to a known technology.

Fifth, if $m \geq n$, values of $S^i_j$ and $P_j$ need not yield positive values for $-U_i/\lambda$, the marginal willingness to pay to reduce intensity of the ith symptom. Of course, in the simple case where $m = n = 1$, the only requirement is that $-P_1/S^1_1 > 0$. If $m = n = 2$, a case considered in the empirical work presented in Section IV, values of $-U_1/\lambda$ and $-U_2/\lambda$ both will be positive only if $(S^1_1/S^1_2) \gtrless (P_1/P_2) \lessgtr (S^2_1/S^2_2)$. If $V_1$ and $V_2$ are not chosen such that their marginal rates of technical substitution bracket their price ratio, then it is possible to reduce intensity of one symptom without increasing intensity of the other and without spending more on symptom reduction.

Sixth, complications arise in expressing symptom and air pollution values in situations where some or all of the $V_j$ are sources of direct utility, another form of joint production. This problem is important (and it is encountered in the empirical work presented in Section IV) because of the difficulty in identifying private goods that are purchased but do not enter the utility function. To illustrate, assume that $m = 2$, $n = 1$ and that $V_2$ but not $V_1$ is a source of both direct positive utility and symptom relief. $WTP_\alpha$ still would equal $-(P_1S^1_\alpha/S^1_1)$ and therefore could be calculated without knowing values for marginal utility terms. If consumption of $V_2$, however, was used as a basis for this calculation, the simple formula $-(P_2S^1_\alpha/S^1_2)$ would overestimate $WTP_\alpha$ by an amount equal to $-(U_2S^1_\alpha/\lambda S^1_2)$ where $U_2$ denotes marginal utility of $V_2$ ($U_2 > 0$). When m and n take arbitrary values the situation is more complex, but in general nonmarket commodity and public good values can be determined only if the number of private goods which do not enter the utility function is at least as great as the number of final commodities. Even if this condition is not met, however, it is possible in some cases to determine whether the value of nonmarket commodities and public goods is over- or underestimated.[4] Each of these six issues is treated in the empirical work reported in Section IV. Although $m = n = 2$ and relevant marginal rates of technical substitution generally bracket input price ratios, statistical tests cannot reject the hypothesis that the technology matrix has rank one. After aggregating symptoms into one broad category, $m > n$ $(2 > 1)$, and first order conditions constrain the marginal rate of technical substitution to equal the price ratio. Failure to reject the constraint confirms that behavior is consistent with the model's predictions; nevertheless the likely possibility that both private good inputs are direct sources of utility suggests that the model's value estimates should be interpreted as lower bounds.

## III. Data

Data used to implement the household production approach were obtained from a sample of 226 residents of two Los Angeles area communities. Each respondent previously had participated in a study of chronic obstructive respiratory disease (Detels et al. 1979, 1981). Key aspects of this sample are: (1) persons with physician diagnosed chronic respiratory ailments deliberately are overrepresented (76 respondents suffered from such diseases), (2) 50 additional respondents with self-reported chronic cough or chronic shortness of breath are included, (3) 151 respondents lived in Glendora, a community with high oxidant air pollution, and 75 respondents lived in Burbank, a community with oxidant pollution levels more like other urbanized areas in the U.S. but with high levels of carbon monoxide, (4) all respondents either were nonsmokers or former smokers who had not smoked in at least two years, and (5) all respondents were household heads with full-time jobs (defined as at least 1,600 hours of work annually).

Professionally trained interviewers contacted respondents several times over a 17 month period beginning in July 1985. The first contact involved administration of an extensive baseline questionnaire in the respondent's home. Subsequent interviews were conducted by telephone.[5] Including the baseline interview, the number of contacts with each respondent varied from three to six with an average number of contacts per respondent of just over five. Of the 1147 total contacts (≅ 226 x 5), 644 were with respiratory impaired subjects (i.e., those either with physician-diagnosed or self-reported chronic respiratory ailments) and 503 were with respondents having normal respiratory function.

Initial baseline interviews measured four groups of variables: (1) long term health status, (2) recently experienced health symptoms, (3) use of private goods and activities that might reduce symptom intensity, and (4) socioeconomic/demographic and work environment characteristics. Telephone follow-up interviews inquired further about health symptoms and use of particular private goods. Long term health status was measured in two ways. First, respondents indicated whether a physician ever had diagnosed asthma (ASTHMA), chronic bronchitis (BRONCH), or other chronic respiratory disease such as emphysema, tuberculosis, or lung cancer. Second, they stated whether they experience chronic shortness of breath or wheezing (SHRTWHZ) and/or regularly cough up phlegm, sputum, or mucous (FLEMCO). Respondents also indicated whether a physician ever had diagnosed hay fever (HAYFEV); however, this condition was not treated as indicative of a chronic respiratory impairment.

Both background and follow-up instruments also asked which, if any, of 26 health symptoms were experienced in the two days prior to the interview. Symptoms initially were aggregated into two categories defined as: (1) chest and throat symptoms and (2) all other symptoms.[6] Aggregation to two categories reduces the number of household produced final goods (n) considered; however, assigning particular symptoms to these categories admittedly is somewhat arbitrary. Yet, the classification scheme selected permits focus on a group of symptoms in which there is current policy interest. Chest and throat symptoms identified have been linked to ambient ozone exposure (see Gerking et al. 1984, for a survey of the evidence) and federal standards for this air pollutant currently are under review. Moreover, multivariate tobit turns out to be a natural estimation method and aggregating symptoms into two categories permits a reduction in computation burden. Dickie et al. (1987(a)) report that respondents with chronic respiratory impairments experienced each of the 26 individual symptoms more often than respondents with normal respiratory function. This outcome is reflected in Table 1 which tabulates frequency distributions of the total number of chest and throat and other symptoms reported by respondents in the two subsamples.[7]

In the empirical work reported in Section IV, data on the number of symptoms reported are assumed to be built up from unobserved latent variables measuring symptom intensity. As intensity of a particular symptom such as cough rises above a threshold, the individual reports having experienced it; otherwise he does not. Thus, the frequency distribution tabulated in Table 1 merely reflects the number of symptoms that crossed the intensity threshold in the two days prior to the interview.

Private goods used to estimate symptom production functions include durable goods which may relieve symptoms by reducing exposure to air pollution. When asked during the baseline interview whether they changed their activities at all when the air was smoggy, half the respondents in the impaired group and 42 percent of the respondents in the normal group reported that they tried to stay indoors and/or run their air conditioners more in an attempt to avoid the pollution. The effectiveness of such a strategy depends on the quality of the indoor air, which in turn depends partly on whether the respondent has and uses the following private goods: (1) central air conditioning in the home (ACCEN), (2) an air purifying system in the home, and (3) a fuel other than natural gas for cooking (NOTGASCK).[8] Similarly, a respondent who has and used air conditioning in the automobile (ACCAR) might reduce exposure to pollution, particularly when driving or idling in traffic. Each of these private goods may provide direct utility in addition to reducing exposure to pollution. Air conditioners, for example, may provide not only relief from symptoms but also cooling services that yield direct satisfaction. This problem is discussed further in Section V.

Socioeconomic/demographic variables measured whether the respondent lived in Burbank or Glendora (BURB) as well as years of age (AGE), gender, race (white or nonwhite), marital status, and household income. Also, respondents were asked whether they were exposed to toxic fumes or dust while at work (EXPWORK).

Finally, each contact with a respondent was matched to measures of ambient air pollution concentrations, humidity, and temperature for that day. Air monitoring stations used are those nearest to residences of respondents in each of the two communities. Measures were obtained of the six criteria pollutants for which national ambient air quality standards have been established: carbon monoxide (CO), nitrogen dioxide (NO2), ozone (O3), sulfur dioxide (SO2), lead and total suspended particulates. Readings for lead and particulates, however, only were available for about ten percent of the days during the study period, forcing exclusion of those pollutants from empirical work. Each of the remaining four pollutants were measured as maximum daily one-hour ambient concentrations. Maxima are used because epidemiological and medical evidence suggests that acute symptoms may be more closely related to peak than to average pollution concentrations. The air pollution variables entered then, are averages of one hour maxima on the two days prior to the interview so as to conform with the measurement of symptoms.[9] Temperature and relative humidity data similarly were averaged across two day periods.

## IV. Estimates of Household Symptom Technology

This section reports estimated production functions, hypothesis tests, and estimated values of public goods and nonmarket commodities. A bivariate tobit model with variance components was developed to account for: (1) probable correlation of disturbances across production functions, (2) censoring of reported symptoms at zero, and (3) repeated observations of the same individuals at different times.[10] Both tobit and variance components models frequently are applied; however, as discussed by Maddala (1987), there have been few applications of tobit with variance components to panel data.

Empirical estimates of household production functions for health also have been obtained by Rosenzweig and Schultz (1983)[11] and variance components models have been applied to health production by Wilcox-Gök (1983, 1985)[12]; however, neither of these investigators focus on valuing nonmarket commodities and public goods. Rosenzweig and Schultz consider birthweight rather than symptoms and Wilcox-Gök examines days missed from usual activities due to illness or injury and visits to certain health care facilities. Although the dependent variables used by Wilcox-Gök would appear to be correlated and censored at zero, the estimation procedures employed by Wilcox-Gök did not correct for either problem. In contrast, the bivariate tobit model presented below allows for both censoring and cross-equation error correlation.

The symptom production functions are specified as

$$S^i_{ht} = \begin{cases} X'_{ht}\beta_i + \varepsilon_{iht} & \text{if } X'_{ht}\beta_i + \varepsilon_{iht} > 0 \\ 0 & \text{otherwise} \end{cases} \qquad (7)$$

$$i = 1,\ 2.$$

In equation (7), i denotes type of symptom (chest and throat = 1, other = 2), h denotes respondent, and t denotes time; $S^i_{ht}$ represents the number of symptoms reported and $X_{ht}$ is a vector including explanatory variables such as measures of health capital, private goods, and air pollutants.

Random disturbances consist of the sum of a transitory component and a permanent component common to both production functions

$$\varepsilon_{iht} = \mu_h + \nu_{iht} \qquad i = 1,\ 2 \qquad (8)$$

The transitory error components, $\nu_{iht}$, capture unmeasured influences that vary over individuals, symptoms, or time. The permanent error component, $\mu_h$, varies only over individuals, capturing unmeasured individual specific influences that persist over time. The assumption that the same permanent component enters both production functions results in computational savings and is at least plausible, since the same individual produces both categories of symptoms.

Permanent components are assumed normally and independently distributed with mean zero and variance $\sigma_\mu^2$. Transitory components are assumed normally and independently distributed, conditional on the permanent component, with mean zero and variance $\sigma_i^2$, $i = 1, 2$. Despite the common permanent component, the correlation coefficient between the two symptom classes in the same time period, $\sigma_\mu^2/(\sigma_\mu^2 + \sigma_1^2)^{\frac{1}{2}}(\sigma_\mu^2 + \sigma_2^2)^{\frac{1}{2}}$, is distinct from the correlation coefficient between the same symptom class at different times, $\sigma_\mu^2/(\sigma_\mu^2 + \sigma_i^2)$, $i = 1, 2$.

Let $F_{iht}$ and $f_{iht}$ represent, respectively, the normal distribution and density functions evaluated at $(S_{ht}^i - X_{ht}'\beta_i - \mu_h)/\sigma_i$, conditional on $\mu_h$. The log-likelihood function is

$$L = \Sigma_h \ln \int \Pi_i [\prod_{S_{ht}^i=0} F_{iht} \cdot \prod_{S_{ht}^i>0} f_{iht}] g(\mu) d\mu \tag{9}$$

where $g(\cdot)$ is the normal density.[13]

An alternative to the variance components or random effects model is the fixed effects model in which the $\mu_h$ are treated as fixed constants rather than as random variables. Two arguments can be made in favor of the random effects specification of the symptom production model.[14]

First, treating the $\mu_h$ as constants subsumes the effects of all individual specific, time invariant variables into the fixed effects. Since the private goods measured in the data are fixed during the sampling period, using the fixed effects model would make it impossible to identify the production function parameters $(S_j^i)$ necessary to estimate values for reductions in symptoms and air pollutants. Similarly, estimating the separate effects for the various chronic health impairment variables is of some interest, but these effects could not be distinguished from the $\mu_h$ in the fixed effects specification.

The second argument in favor of random effects rests on the inconsistency of the fixed effects tobit estimator. The individual effects $\mu_h$ cannot be estimated consistently for a small number of time periods even as the number of individuals increases without bound. Intuitively, each individual brings to the sample a distinct $\mu_h$, with the result that increasing the number of individuals fails to increase the information available to estimate the $\mu_h$. In many nonlinear models, including tobit, fixed effects estimators for the remaining parameters cannot be derived independently of the $\mu_h$, so that the entire set of parameters is estimated inconsistently. By contrast, the random effects model attempts to estimate only the mean and variance of the $\mu_h$ rather than the individual effects themselves and thus can estimate the slope coefficients of the model consistently.

While these arguments present a compelling case for the random effects model, biased estimation can result because the model ignores the correlation that may exist between the explanatory variables and the permanent error component (see, e.g., Mundlak 1978). For example, if an individual knows his own $\mu_h$, utility maximization would imply that his choice of private goods depends on $\mu_h$. A solution to this problem proposed for probit models by Chamberlain (1980) is to specify $\mu_h$ as a linear function of the individual's explanatory variables plus an orthogonal residual: $\mu_h = X_h'\Pi + \eta_h$, where $X_h'$ includes the individual's entire time series of observations on explanatory variables. This auxiliary regression then could be substituted for $\mu_h$ in the specification of the symptom production functions, and the likelihood derived by integrating over the density of $\eta$ rather than the density of $\mu$. But owing to the lack of temporal variation in all explanatory variables except the measures of pollution and weather, the substitution would produce collinearity in the matrix of explanatory variables as each time-invariant variable in the auxiliary regression above would be perfectly collinear with its counterpart already included in the model specification. As a consequence, Chamberlain's approach was not pursued.

An alternative approach to correct for correlation between covariates and errors is analogous to the two stage least squares procedure employed by Rosenzweig and Schultz in their previously cited birthweight study. In the first stage, reduced form probit demand equations for each of four private goods (ACHOME, ACCAR, APHOME, NOTGASCK) are estimated.[15] In the second stage, predicted probabilities from the reduced form probits were to be used as instruments for private goods in the tobit symptom production function models, but explanatory power of the reduced form probit equations was very poor. In half of the equations for each subsample the null hypothesis that all slope coefficients jointly are zero could not be rejected at the 5 percent level and in all equations key variables such as household income had insignificant and often wrongly signed coefficients. Another problem is the absence of private good price data specific to each respondent. The original survey materials requested these data but after pretesting, this series of questions was dropped because many respondents often made purchases jointly with a house or car and were unable to provide even an approximate answer. As a consequence, two-stage estimation was not pursued further with the likely outcome that estimates of willingness to pay for nonmarket commodities and public goods may have a downward bias.

Tables 2 and 3 present illustrative symptom production function estimates for impaired and normal subsamples. Equations presented are representative of a somewhat broader range of alternative specifications available from the authors on request. The overall explanatory power of the model was evaluated by testing the null hypothesis that all estimated coefficients (excepting the constant terms) jointly are zero. A likelihood ratio tests rejects this hypothesis for both subsamples at significance levels less than one percent. Also, estimates of the individual specific error components, denoted $\sigma_{\mu}$, have large asymptotic t-statistics which confirms persistence of unobserved personal characteristics that affect symptoms.

Table 2 shows that chronic health ailments and hay fever are positively related to symptom occurrence among members of the impaired group. Coefficients of ASTHMA, BRONCH, SHRTWHZ, and HAYFEV are positive in equations for both chest and throat and other symptoms and have associated asymptotic t-statistics that range from 2.1 to 7.6. The coefficient of FLEMCO is positive and significantly different from zero at conventional levels in the chest and throat equation, but its asymptotic t-statistic is less than unity in the equation for other symptoms. The coefficient of AGE was not significantly different from zero in either equation and the EXPWORK variable was excluded because of convergence problems with the bivariate tobit algorithm.[16] Variables measuring gender, race, and marital status never were included in the analysis because 92 percent of the impaired respondents were male, 100 percent were white, and 90 percent were married. Residents of Burbank experience chest and throat symptoms with less frequency than do residents of Glendora. Of course, many possible factors could explain this outcome; however, Burbank has had a less severe long term ambient ozone pollution problem than Glendora. For example, in 1986 average one day hourly maximum ozone readings in Burbank and Glendora were 8.7 pphm and 10.2 pphm, respectively, and a similar difference in ozone readings has persisted at least since 1983.

With respect to private and public inputs to the symptom production functions, the coefficient of ACCAR is negative and significantly different from zero at the 10 percent level using a one tail test in the other symptoms equation, while the coefficient of ACCEN is negative and significantly different from zero at the 5 percent level using a one tail test in both equations. Results from estimated equations not presented reveal that NOTGASCK and use of air purification at home never are significant determinants of symptoms in the impaired subsample. Also, O3, CO, and NO2 exert insignificant influences on occurrence of both types of symptoms. When four air pollution variables were entered, collinearity between them appeared to prevent the maximum likelihood algorithm from converging. Consequently, SO2 was arbitrarily excluded from the specification presented and the three air pollution measures included as covariates should be interpreted as broader indices of ambient pollutant concentrations. Variables measuring temperature and humidity were excluded from the Table 2 specification; but in equations not reported their coefficients never were significantly different from zero.

Table 3 presents corresponding symptom production estimates for the subsample with normal respiratory function. HAYFEV is the only health status variable entered because ASTHMA, BRONCH, SHRTWZ, and FLEMCO were used to define the impaired subsample. Coefficients of HAYFEV are positive in equations for both chest and throat and other symptoms and have t-statistics of 1.61 and 1.87, respectively. Coefficients of BURB are negative; but in contrast to impaired subsample results, they are not significantly different from zero at conventional levels. AGE and EXPWORK enter positively and their coefficients differ significantly from zero at 2½ percent in the other symptoms equation. Among private goods entering the production functions, coefficients of APHOME and ACHOME never were significantly different from zero at conventional levels, and these variables are excluded from the specification in Table 3. Use of air conditioning in an automobile reduced chest and throat symptom occurrences and cooking with a fuel other than natural gas (marginally) reduces other symptoms. Variables measuring gender, race, and marital status again were not considered as the normal subsample was 94 percent male, 99 percent white, and 88 percent married. In the normal subsample, collinearity and algorithm convergence problems again limited the number of air pollution variables that could be entered in the same equation. As shown in Table 3, O3, CO, and NO2 coefficients had associated t-statistics of 1.16 or smaller. Temperature and humidity variables are excluded from the specification shown in Table 3. In alternative specifications not reported, coefficients of these variables never were significantly different from zero in alternative equations not reported.

Three pieces of information are required to use the estimates in Tables 2 and 3 in the calculation of values for reductions in symptoms and air pollutants: (1) marginal effects of air pollutants on symptoms, (2) marginal effects of private goods on symptoms, and (3) prices of private goods. Marginal products were defined as the effect of a small change in a good on the expected number of symptoms. Computational formulae were developed extending results for the tobit model (see McDonald and Moffit 1980) to the present context which allows for variance components error structure. However, because private goods are measured as dummy variables and, therefore, cannot be continuously varied, incremental, rather than marginal, products are used.

The final elements needed to compute value estimates are the prices of private goods. Dealers of these goods in the Burbank and Glendora areas were contacted for estimates of initial investment required to purchase the goods, average length of life, scrap value (if any), and fuel expense. After deducting the present scrap value from the initial investment, the net initial investment was amortized over the expected length of years of life. Adding annual fuel expense yields an estimate (or range of estimates) of annual user cost of the private good. The annual costs then were converted to two-day costs to match the survey data.[17] The dependent variables used in the estimated equations do not distinguish between one- and two-day occurrences of symptoms, but approximately one-half of the occurrences were reported as two day occurrences. As a consequence, the value estimates obtained were divided by 1.5 to convert to daily values.

Two tests were performed prior to estimating values of symptom and air pollution reduction. First, calculations were made for both normal and impaired subsamples to ensure that relevant ratios of incremental products of private goods in reducing symptoms bracketed the corresponding price ratio. Recall from the discussion in Section II that this condition guarantees that value estimates for reducing both types of symptoms are positive. A problem in making this calculation is that estimates of incremental rates of technical substitution vary across individuals (incremental products are functions of individual characteristics), but no respondent specific price information is available. As just indicated, dealers in Glendora provided the basis for a plausible range of prices to be constructed for each good. If midpoints of relevant price ranges are used together with incremental rates of technical substitution taken from Tables 2 and 3, the bracketing condition is met for all 100 respondents in the normal subsample and 117 of 126 respondents in the impaired subsample. Of course, alternative price ratios selected from this range meet the bracketing condition for different numbers of respondents.

Second, possible singularity of the symptom technology matrix was analyzed using a Wald test (see Judge et al. 1985, p. 215 for details).[18] In the context of estimates in Tables 2 and 3, the distribution of the test statistic ($\lambda$) is difficult to evaluate because relevant derivatives are functions of covariate values and specific to individual respondents. However, if derivatives are evaluated in terms of the underlying latent variable model, they can be expressed in terms of parameters only and $\lambda$ is distributed as $\chi^2$ with 1 degree of freedom. Adopting this simpler approach, p-values for the Wald test statistic are large: $p = .742$ for the impaired subsample equations and $p = .610$ for the normal subsample equations.[19] As a consequence, the null hypothesis of singularity of the symptom technology matrix is not rejected at conventional levels. This result suggests that in both subsamples, there does not appear to be an independent technology for reducing the two types of symptoms, budget constraints are nondifferentiable, and separate value estimates for chest and throat and other symptoms should not be calculated.

A common value for reducing chest and throat and other symptoms still can be obtained by aggregating the two categories and re-estimating production functions in a univariate tobit framework. Table 4 shows results based on using the same covariates as those reported in Tables 2 and 3 and retaining the variance components error structure. The Table 4 equations also make use of a constraint requiring that if $m > n = 1$, the marginal rate of technical substitution must equal the input price ratio to insure that values of marginal willingness to pay to avoid a symptom must be identical no matter which private good is used as the basis for the calculation. In the case where $m = 2$ and $n = 1$, as discussed in Section II, this single value is $-U_1/\lambda = -(P_1/S_1^1) = -(P_2/S_2^1)$. In the impaired subsample, the restriction can be tested under the null hypothesis, $H_0 : \beta_{ACCAR} = (P_{ACCAR}/P_{ACHOME})\beta_{ACHOME}$, where the $\beta_i$ are coefficients of ACCAR and ACHOME in the latent model and the $P_i$ are midpoints from the estimated range of two day prices for the private goods. In corresponding notation, the null hypothesis to test in the normal subsample is, $H_0 : \beta_{ACCAR} = (P_{ACCAR}/P_{NOTGASCK})\beta_{NOTGASCK}$. Both hypotheses are tested against the alternative that coefficients of private goods are unconstrained parameters, using a likelihood ratio test.

P-values for the parameter restrictions are comparatively large; $P = .623$ in the impaired subsample and $P = .562$ in the normal subsample. Thus, the above null hypotheses are not rejected at conventional significance levels. This result supports a critical implication of the previously presented household production model, namely that individuals equate marginal rates of technical substitution in production with relevant price ratios. Moreover, coefficients of private good variables defined under the null hypotheses for the two subsamples have t-statistics exceeding two in absolute value. Performance of remaining variables is roughly comparable to the bivariate tobit estimates. A notable exception, however, is that in the normal subsample univariate tobit estimates, coefficients of O3 and NO2 are positive with t-statistics exceeding 1.6. This outcome suggests that persons with normal respiratory function tend to experience more symptoms when air pollution levels are high, whereas those with impaired respiratory function experience symptoms with such regularity that there is no clear relationship to fluctuations in air quality. Intensity of particular symptoms may be greater in both subsamples when pollution levels are high, but this aspect is not directly measured.

Table 5 presents estimates of marginal willingness to pay to avoid symptoms and to reduce two air pollutants. Unconditional values of relieving symptoms and reducing air pollution are calculated for each respondent from observed univariate tobit models. Table 5 reports the mean, median, and range of respondents' marginal willingness to pay to eliminate one health symptom for one day as well as mean marginal willingness to pay to reduce air pollutants by one unit for one day for the normal subsample. Symptom reduction values range from $0.81 to $1.90 in the impaired subsample and from $0.49 to $1.22 in the normal subsample with means of $1.12 and $0.73 in the two subsamples, respectively.[20] Also, values of willingness to pay to reduce one hour daily maximum levels of O3 and NO2 by one part per ten million are $0.31 and $0.91 in the normal subsample. Corresponding calculations are not reported for the impaired subsample because, as shown in Table 4, coefficients of air pollution variables are not significant at conventional levels.

## V. Conclusion

Willingness to pay values of symptom reduction and air quality improvement just presented should be viewed as illustrative approximations for two reasons. First, private goods used in computing the estimates are likely to be direct sources of utility. Second, symptom experience and private good purchase decisions are likely to be jointly determined. Nevertheless, these estimates still are of interest because aspects of joint production are taken into account. A key finding is that independent technologies for home producing symptoms are difficult to identify, thus greatly limiting the number of individual symptoms for which values can be computed. In fact, the 26 symptoms analyzed here had to be aggregated into a single group before willingness to pay values could be computed.

This outcome appears to have implications for estimating willingness to pay for nonmarket commodities in other contexts. An obvious example concerns previous estimates of willingness to pay to avoid health symptoms. Berger et al. (1987) report one day willingness to pay values for eliminating each of seven minor health symptoms, such as stuffed up sinuses, cough, headache and heavy drowsiness that range from $27 per day to $142 per day. Green et al. (1978) present estimates of willingness to pay to avoid similarly defined symptoms ranging from $26 per day to $79 per day. In both studies, however, willingness to pay estimates were obtained symptom by symptom in a contingent valuation framework that ignores whether independent technologies are available to produce each. Thus, respondents simply may have lumped total willingness to pay for broader health concerns onto particular symptoms. Some respondents may also have inadvertently stated their willingness to pay to avoid symptoms for periods longer than one day.

Another example relates to emerging research aimed at splitting willingness to pay to reduce air pollution into health, visibility, and possibly other components. From a policy standpoint, this line of inquiry is important because the Clean Air Act and its subsequent amendments focus primarily on health and give less weight to other reasons why people may want lower air pollution levels. Analyzing location choice within metropolitan areas, for example, may not provide enough information to decompose total willingness to pay into desired components. Instead, survey procedures must be designed in which respondents are either reminded of independent technologies that can be used to home produce air pollution related goods or else confronted with believable hypothetical situations that allow one good to vary while others are held constant.

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## FOOTNOTES

[1] Another, possibly troublesome, aspect of joint production occurs if some or all elements of V are arguments in the utility function. This complication is discussed momentarily.

[2] Hori identifies three sources of nondifferentiability of the budget surface under joint production. The first occurs if the number of private goods is less than the number of commodities. The second arises because of nonnegativity restrictions on the private goods. This is not treated directly in the present paper, but if each private good is purchased in positive quantities, the chosen commodity bundle will not lie at the second type of kink. Hori's third cause of nondifferentiability implies linear dependence among the rows of the technology matrix, a possibility considered below.

[3] Notice that this point on aggregation may apply to other valuation methods as well. Using contingent valuation surveys, for example, Green et al. (1978) and Berger et al. (1987) obtained value estimates of several specific symptoms; however, issues relating to existence of independent symptom technologies never was faced. Future contingent valuation surveys may do well to consider this point prior to eliciting estimates of willingness to pay.

[4] For example, suppose $m = n = 2$ and both private goods are direct sources of utility. If equation (6) is used to solve for the $U_i/\lambda$, then: (1) if the two marginal rates of technical substitution (MRTS) do not bracket the price ratio, then the value of the commodity whose

MRTS is closer in magnitude to the price ratio will be overestimated, while the value of the other commodity will be underestimated; (2) if the two MRTS values do bracket the price ratio, then the value of either one or both of the commodities will be overestimated; and (3) in no case will the value of both commodities be underestimated.

[5] Both questionnaires are presented and extensively discussed in Volume II of Dickie et al. (1987(b)).

[6] Chest and throat symptoms include (1) cough, (2) throat irritation, (3) husky voice, (4) phlegm, sputum or mucous, (5) chest tightness, (6) could not take a deep breath, (7) pain on deep respiration, (8) out of breath easily, (9) breathing sounds wheezing or whistling. Other symptoms are (1) eye irritation, (2) could not see as well as usual, (3) eyes sensitive to bright light, (4) ringing in ears (5) pain in ears, (6) sinus pain, (7) nosebleed, (8) dry and painful nose, (9) runny nose, (10) fast heartbeat at rest, (11) tired easily, (12) faintness or dizziness, (13) felt spaced out or disoriented, (14) headache, (15) chills or fever, (16) nausea, and (17) swollen glands.

[7] An alternative to counting the number of different symptoms experienced in the two days prior to the interview would be to consider the number of symptom/days experienced. Both approaches were used to construct empirical estimates; however, to save space, only those based on counts of different symptoms are reported. Both approaches yield virtually identical value estimates for symptom and air pollution reduction.

[8] Cooking with a fuel other than natural gas reduces exposure because gas stoves emit nitrogen dioxide.

[9] The equations also were estimated after defining the pollution variables as the largest of the one hour maxima on the two days; similar results were obtained.

[10] Although there is a linear relationship between the latent dependent variables and the private goods in the tobit model, the relationship between the observed dependent variables and the private goods has the usual properties of a production function. The expected number of symptoms is decreasing and convex (nonstrictly) in the private goods.

[11] Rosenzweig and Schultz also initially specify their production functions in translog form and then test whether restrictions to CES and Cobb-Douglas forms are justified. This type of analysis is not pursued here as most of the covariates used are 0-1 dummy variables. Squaring these variables does not alter their values. Interaction variables of course, still could be computed.

[12] Wilcox-Gök used variance components to control for family-specific effects in pooled sibling data rather than for individual-specific effects in pooled cross section-time series data.

[13] The tobit coefficients and variances of the model are estimated by maximizing the likelihood function using the method of Berndt, Hall, Hall, and Hausman (1974). The score vectors are specified analytically and the information matrix is approximated numerically using the summed outer products of the score vectors. Starting values for the coefficients and the standard deviations of the transitory error components were obtained from two independent tobit regressions with no permanent error component. In preliminary runs a starting value of unity was used for the standard deviation of the permanent error component, but the starting value was

adjusted to 1.5 after the initial estimate was consistently greater than one.

[14] The following discussion draws heavily on Hsiao (1986) and Maddala (1987).

[15] Covariates in the reduced form regressions are: ASTHMA, BRONCH, FLEMCO, SHRTWZ, HAYFEV, BURB, AGE, EXPWORK, years of education, number of dependents, household income, and an occupation dummy variable measuring whether respondent is a blue collar worker.

[16] In the impaired subsample, inclusion of EXPWORK frequently caused the bivariate tobit algorithm to fail to converge. This problem arose in the specification presented in Table 2; consequently the EXPWORK variable was excluded.

[17] The estimated two-day prices are: \$2.34 for ACCEN, \$1.00 for ACCAR, \$0.80 for NOTGASCK. The discount rate was assumed to be 5 percent. For further details of the procedure used to estimate prices, see Dickie et al. (1987(a)).

[18] The Wald test was chosen because its test statistic can be computed using only the unconstrained estimates. Since the likelihood and constraint functions both are nonlinear, re-estimating the model with the constraint imposed would be considerably more difficult than computing the Wald test statistic. Gregory and Veall (1985) identified a problem with Wald tests of nonlinear restrictions: changing the restriction into a form that is algebraically equivalent under the null hypothesis will change the p-value of the test. To check for this problem, the constraint was tested in two forms. The first, reported in the text, is $H_0 : S^1_1 S^2_2 - S^1_2 S^2_1 = 0$. The second is $S^1_1/S^1_2 - S^2_1/S^2_2 = 0$. In all cases both tests yielded nearly identical p-values.

[19] In other estimates of symptom production functions not reported here, corresponding p-values also are large, almost always exceeding .25 and sometimes the .80-.90 range.

[20] For comparison purposes, mean values also were estimated at subsample means of all explanatory variables. Results differ little with means computed over respondents. Evaluated at subsample means, willingness to pay to eliminate one symptom for one day is \$1.05 in the impaired subsample and \$0.70 in the normal subsample.

TABLE 1.--FREQUENCY DISTRIBUTIONS OF SYMPTOMS BY SUBSAMPLE

| | Number of Chest and Throat Symptoms Experienced in Past Two Days | | Number of Other Symptoms Experienced In Past Two Days | |
|---|---|---|---|---|
| | Impaired | Normal | Impaired | Normal |
| 0 | 351 | 408 | 257 | 338 |
| 1 | 84 | 41 | 123 | 79 |
| 2 | 64 | 18 | 85 | 42 |
| 3 | 48 | 15 | 73 | 18 |
| 4 | 37 | 9 | 45 | 12 |
| 5 | 26 | 4 | 28 | 5 |
| 6 | 16 | 6 | 14 | 6 |
| 7 | 8 | 2 | 9 | 2 |
| 8 | 8 | 0 | 4 | 1 |
| 9 | 2 | 0 | 2 | 0 |
| 10 | 0 | 0 | 1 | 0 |
| 11 | 0 | 0 | 1 | 0 |
| 12 | 0 | 0 | 2 | 1 |
| 13 | 0 | 0 | 0 | 0 |
| 14 | 0 | 0 | 0 | 0 |
| 15 | 0 | 0 | 0 | 0 |
| 16 | 0 | 0 | 0 | 0 |
| 17 | 0 | 0 | 0 | 0 |
| Sample Mean | 1.348 | 0.453 | 1.668 | 0.692 |

## TABLE 2.--BIVARIATE TOBIT SYMPTOM PRODUCTION FUNCTION ESTIMATES: IMPAIRED SUBSAMPLE[a]

| | Chest and Throat Symptoms | Other Symptoms |
|---|---|---|
| CONSTANT | -3.085<br>(-3.035) | -2.043<br>(-2.125) |
| ASTHMA | 0.8425<br>(2.328) | 0.6724<br>(1.851) |
| BRONCH | 3.774<br>(7.663) | 2.936<br>(6.668) |
| SHRTWHZ | 1.494<br>(3.683) | 1.235<br>(3.428) |
| FLEMCO | 1.458<br>(4.038) | 0.2526<br>(0.8558) |
| HAYFEV | 1.110<br>(3.509) | 0.6613<br>(2.365) |
| BURB | -1.431<br>(-2.728) | -0.7330<br>(-1.539) |
| AGE | 0.2986<br>(0.1596) | 2.042<br>(1.177) |
| EXPWORK | ---[b] | ---[b] |
| ACCAR | -0.3485<br>(-0.8885) | -0.4395<br>(-1.364) |
| ACCEN | -1.9961<br>(-2.834) | -0.6291<br>(-1.829) |
| O3 | -0.1672<br>(-0.5638) | 0.1252<br>(-.4475) |
| CO | 1.279<br>(1.259) | -0.06285<br>(-0.06356) |
| NO2 | 0.5475<br>(0.7744) | 0.6384<br>(0.9282) |
| $\sigma_\nu$ | 2.617<br>(17.70) | 2.454<br>(20.81) |
| $\sigma_\mu$ | 1.827<br>(21.17) | |
| Chi-Square[c] | 148.7 | |
| P-Value for Wald Test | 0.742 | |
| Number of Iterations[d] | 21 | |

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[a] The dependent variables are the numbers of symptoms reported in the "chest and throat" category and in the "other" category. Asymptotic t-ratios are in parentheses. AGE is measured in centuries, CO in parts per hundred thousand, and O3 and NO2 in parts per ten million. All remaining explanatory variables are dummies. Note the long term health status dummies do not represent mutually exclusive categories.

[b] Omitted due to convergence problems.

[c] The chi-square test statistic is $-2\ln\lambda$, where $\lambda$ is the likelihood ratio, for a test of the null hypothesis that the slope coefficients in both production functions are all zero.

[d] The convergence criterion is 0.5 for the gradient-weighted inverse Hessian.

## TABLE 3.--BIVARIATE TOBIT SYMPTOM PRODUCTION FUNCTION ESTIMATES: NORMAL SUBSAMPLE[a]

| | Chest and Throat Symptoms | Other Symptoms |
|---|---|---|
| CONSTANT | -5.789<br>(-2.157) | -5.479<br>(-2.790) |
| HAYFEV | 2.316<br>(1.614) | 1.461<br>(1.871) |
| BURB | -1.388<br>(-1.180) | -0.6248<br>(-0.8470) |
| AGE | 4.143<br>(0.7873) | 7.075<br>(2.091) |
| EXPWORK | 0.8707<br>(1.157) | 1.329<br>(2.297) |
| ACCAR | -1.949<br>(-2.905) | -0.6705<br>(-1.057) |
| NOTGASCK | -0.4613<br>(-0.6312) | -0.8565<br>(-1.594) |
| O3 | 0.2757<br>(0.5867) | 0.3592<br>(0.9674) |
| CO | 0.1788<br>(0.07729) | -0.07200<br>(-0.05241) |
| NO2 | 1.841<br>(1.162) | 1.069<br>(1.127) |
| $\sigma_\nu$ | 3.204<br>(10.15) | 2.435<br>(11.31) |
| $\sigma_\mu$ | 1.828<br>(10.44) | |
| Chi-Square[b] | 69.81 | |
| P-Value for Wald Test | 0.610 | |
| Number of Iterations[c] | 20 | |

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[a] The dependent variables are the numbers of symptoms reported in the "chest and throat" category and in the "other" category. Asymptotic t-ratios are in parentheses. AGE is measured in centuries, CO in parts per hundred thousand, and O3 and NO2 in parts per ten million. All remaining explanatory variables are dummies.

[b] The chi-square test statistic is $-2\ln\lambda$, where $\lambda$ is the likelihood ratio, for a test of the null hypothesis that the slope coefficients in both production functions are all zero.

[c] The convergence criterion is 0.5 for the gradient-weighted inverse Hessian.

## TABLE 4.--UNIVARIATE TOBIT SYMPTOM PRODUCTION FUNCTION ESTIMATES[a]

| | Impaired Subsample | Normal Subsample |
|---|---|---|
| CONSTANT | -2.253<br>(-1.263) | -6.085<br>(-2.329) |
| ASTHMA | 1.0333<br>(1.953) | |
| BRONCH | 4.649<br>(7.708) | |
| SHRTWHZ | 1.909<br>(3.242) | |
| FLEMCO | 1.769<br>(3.607) | |
| HAYFEV | 1.574<br>(3.137) | 2.216<br>(2.378) |
| BURB | -1.830<br>(-2.927) | -1.623<br>(-1.126) |
| AGE | 1.200<br>(0.4034) | 6.351<br>(1.165) |
| EXPWORK | ---[b] | 1.725<br>(2.039) |
| ACCAR | -0.5900<br>(-2.585) | -1.260<br>(-2.425) |
| O3 | 0.1629<br>(0.4846) | 0.5941<br>(1.616) |
| CO | 1.013<br>(0.8041) | 0.3722<br>(0.2163) |
| NO2 | 0.8930<br>(1.130) | 1.726<br>(1.784) |
| $\sigma_\nu$ | 3.884<br>(37.29) | 3.790<br>(22.47) |
| $\sigma_\mu$ | 2.582<br>(15.84) | 2.516<br>(8.822) |
| Chi-Square[c] | 77.88 | 36.45 |
| P-Value for Parameter Restrictions | 0.623 | 0.562 |
| Number of Iterations[d] | 8 | 5 |

[a] The dependent variable is the total number of symptoms reported. Asymptotic t-ratios are in parentheses. AGE is measured in centuries, CO in parts per hundred thousand, and O3 and NO2 in parts per ten million. All remaining explanatory variables are dummies. Note the long term health status dummies do not represent mutually exclusive categories.

[b] Omitted due to convergence problems.

[c] The chi-square test statistic is $-2\ln\lambda$, where $\lambda$ is the likelihood ratio, for a test of the null hypothesis that the slope coefficients in both production functions are all zero.

[d] The convergence criterion is 0.5 for the gradient-weighted inverse Hessian.

TABLE 5.--MARGINAL WILLINGNESS TO PAY TO RELIEVE SYMPTOMS AND AVOID AIR POLLUTION

| | | Impaired Subsample | | |
|---|---|---|---|---|
| | Symptoms | 03 | NO2 | CO |
| Mean | $1.12 | ---[a] | ---[a] | ---[a] |
| Median | $1.09 | | | |
| Maximum | $1.90 | | | |
| Minimum | $0.81 | | | |

| | | Normal Subsample | | |
|---|---|---|---|---|
| | Symptoms | 03 | NO2 | CO |
| Mean | $0.73 | $0.31[b] | $0.91[b] | ---[a] |
| Median | $0.70 | | | |
| Maximum | $1.22 | | | |
| Minimum | $0.49 | | | |

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[a] Denotes coefficient not significantly different from zero at 10 percent level using one tail test in estimated equations presented in Table 4.

[b] Estimates of willingness to pay for reduced air pollution do not vary across sample members. In the computational ratio, respondent specific information appears both in the numerator and denominator and therefore cancels out.